=> b reg
FILE 'REGISTRY' ENTERED AT 10:45:21 ON 03 MAR 2004

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 2 MAR 2004 HIGHEST RN 657348-90-8
DICTIONARY FILE UPDATES: 2 MAR 2004 HIGHEST RN 657348-90-8

TSCA INFORMATION NOW CURRENT THROUGH JULY 14, 2003

Please note that search-term pricing does apply when conducting SmartSELECT searches.

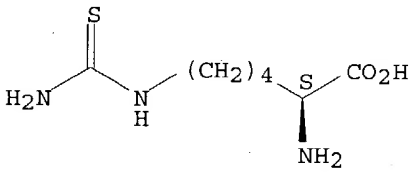
Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. For more information enter HELP PROP at an arrow prompt in the file or refer to the file summary sheet on the web at:
http://www.cas.org/ONLINE/DBSS/registryss.html

=> d ide l160 tot List of all Compounds with their salts that were searched

L160 ANSWER 1 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 625830-82-2 REGISTRY
CN L-Lysine, N6-(aminothioxomethyl)-, dihydrobromide (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H15 N3 O2 S . 2 Br H
SR CA
LC STN Files: CA, CAPLUS
CRN (156719-38-9)

Absolute stereochemistry.



● 2 HBr

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 2 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 346718-49-8 REGISTRY
CN Cellulose, N5-[imino(nitroamino)methyl]-L-ornithine ester (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C6 H13 N5 O4 . x Unspecified
PCT Manual registration, Polyother, Polyother only

SR CA
LC STN Files: CA, CAPLUS, USPATFULL

CM 1

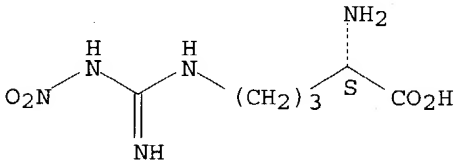
CRN 9004-34-6
CMF Unspecified
CCI PMS, MAN

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

CM 2

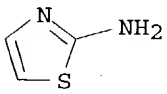
CRN 2149-70-4
CMF C6 H13 N5 O4

Absolute stereochemistry.



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 3 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 330156-58-6 REGISTRY
CN 2-Thiazolamine, dihydrochloride (9CI) (CA INDEX NAME)
MF C3 H4 N2 S . 2 Cl H
SR CA
LC STN Files: CA, CAPLUS
CRN (96-50-4)



●2 HCl

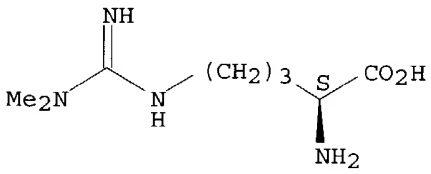
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 4 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 288572-11-2 REGISTRY
CN L-Ornithine, N5-[(dimethylamino)iminomethyl]-, 2-hydroxy-1,2,3-propanetricarboxylate (1:1) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H18 N4 O2 . C6 H8 O7
SR CA
LC STN Files: CA, CAPLUS

CM 1

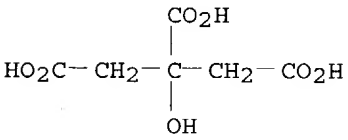
CRN 30315-93-6
CMF C8 H18 N4 O2

Absolute stereochemistry.



CM 2

CRN 77-92-9
CMF C6 H8 O7



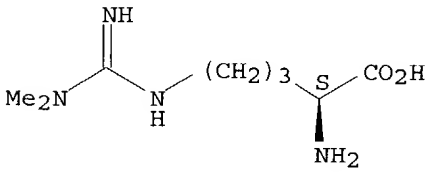
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 5 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 288572-10-1 REGISTRY
CN L-Ornithine, N5-[(dimethylamino)iminomethyl]-, phosphate (1:1) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H18 N4 O2 . H3 O4 P
SR CA
LC STN Files: CA, CAPLUS

CM 1

CRN 30315-93-6
CMF C8 H18 N4 O2

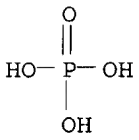
Absolute stereochemistry.



CM 2

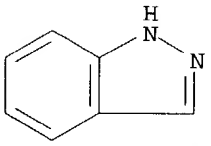
CRN 7664-38-2

CMF H3 O4 P



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 6 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 288160-60-1 REGISTRY
CN 1H-Indazole, lithium salt (9CI) (CA INDEX NAME)
MF C7 H6 N2 . Li
SR CA
LC STN Files: CA, CAPLUS
CRN (271-44-3)



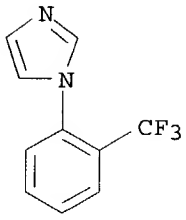
● Li

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 7 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 257626-01-0 REGISTRY
CN 1H-Imidazole, 1-[2-(trifluoromethyl)phenyl]-, mononitrate (9CI) (CA INDEX NAME)
MF C10 H7 F3 N2 . H N O3
SR CA
LC STN Files: CA, CAPLUS, USPATFULL

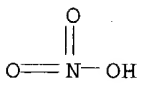
CM 1

CRN 25371-96-4
CMF C10 H7 F3 N2



CM 2

CRN 7697-37-2
CMF H N O3

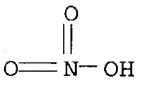


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 8 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 257626-00-9 REGISTRY
CN 1H-Indazole, 7-nitro-, mononitrate (9CI) (CA INDEX NAME)
MF C7 H5 N3 O2 . H N O3
SR CA
LC STN Files: CA, CAPLUS, USPATFULL

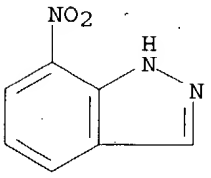
CM 1

CRN 7697-37-2
CMF H N O3



CM 2

CRN 2942-42-9
CMF C7 H5 N3 O2

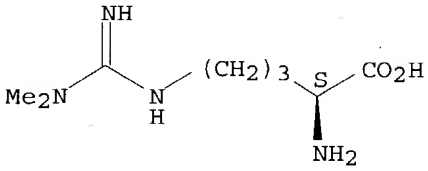


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 9 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 220805-22-1 REGISTRY
CN L-Ornithine, N5-[(dimethylamino)iminomethyl]-, dihydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H18 N4 O2 . 2 Cl H
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, CSCHEM, TOXCENTER

CRN (30315-93-6)

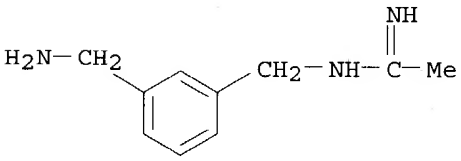
Absolute stereochemistry.



●2 HCl

5 REFERENCES IN FILE CA (1907 TO DATE)
5 REFERENCES IN FILE CAPLUS (1907 TO DATE)

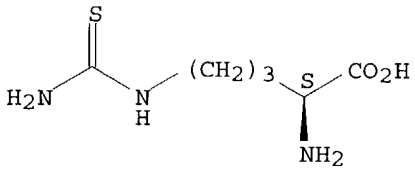
L160 ANSWER 10 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 214358-33-5 REGISTRY
CN Ethanimidamide, N-[[3-(aminomethyl)phenyl]methyl]-, dihydrochloride (9CI) (CA INDEX NAME)
MF C10 H15 N3 . 2 Cl H
SR CAS Client Services
LC STN Files: CHEMCATS
CRN (180001-34-7)



●2 HCl

L160 ANSWER 11 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 212051-53-1 REGISTRY
CN L-Ornithine, N5-(aminothioxomethyl)-, dihydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C6 H13 N3 O2 S . 2 Cl H
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS
CRN (156719-37-8)

Absolute stereochemistry.



● 2 HCl

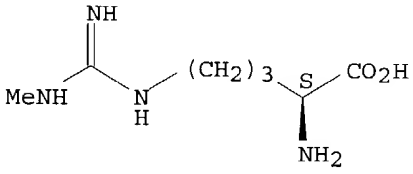
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 12 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 211183-26-5 REGISTRY
CN L-Ornithine, N5-[imino(methylamino)methyl]-, 2-hydroxy-1,2,3-propanetricarboxylate (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H16 N4 O2 . x C6 H8 O7
SR CAS Client Services
LC STN Files: CA, CAPLUS

CM 1

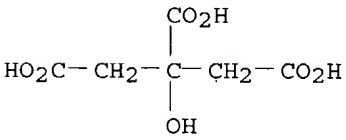
CRN 17035-90-4
CMF C7 H16 N4 O2

Absolute stereochemistry.



CM 2

CRN 77-92-9
CMF C6 H8 O7



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

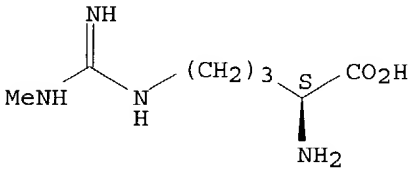
L160 ANSWER 13 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 209913-88-2 REGISTRY
CN L-Ornithine, N5-[imino(methylamino)methyl]-, 2-hydroxy-1,2,3-

propanetricarboxylate (1:1) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H16 N4 O2 . C6 H8 O7
SR CAS Client Services
LC STN Files: CA, CAPLUS

CM 1

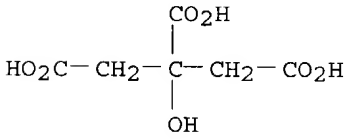
CRN 17035-90-4
CMF C7 H16 N4 O2

Absolute stereochemistry.



CM 2

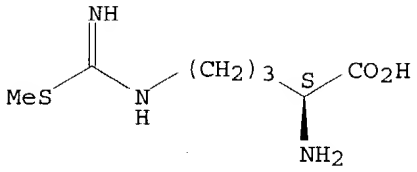
CRN 77-92-9
CMF C6 H8 O7



4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 14 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 209589-59-3 REGISTRY
CN L-Ornithine, N5-[imino(methylthio)methyl]-, dihydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H15 N3 O2 S . 2 Cl H
SR CAS Client Services
LC STN Files: CA, CAPLUS, CHEMCATS, TOXCENTER
CRN (156719-41-4)

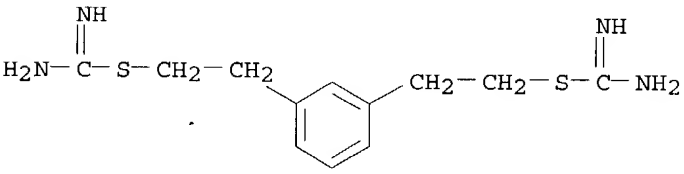
Absolute stereochemistry. Rotation (+).



●2 HCl

5 REFERENCES IN FILE CA (1907 TO DATE)
5 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 15 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 200716-66-1 REGISTRY
CN Carbamimidothioic acid, 1,3-phenylenedi-2,1-ethanediyl ester, dihydrobromide (9CI) (CA INDEX NAME)
MF C12 H18 N4 S2 . 2 Br H
SR CAS Client Services
LC STN Files: CA, CAPLUS
CRN (157254-42-7)



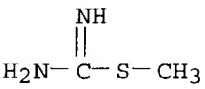
●2 HBr

3 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 16 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 200404-26-8 REGISTRY
CN Carbamimidothioic acid, methyl ester, mono(4-methylbenzenesulfonate) (9CI) (CA INDEX NAME)
MF C7 H8 O3 S . C2 H6 N2 S
SR CAS Client Services
LC STN Files: CA, CAPLUS, CHEMCATS

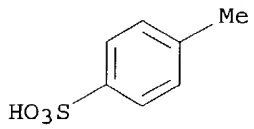
CM 1

CRN 2986-19-8
CMF C2 H6 N2 S



CM 2

CRN 104-15-4
CMF C7 H8 O3 S

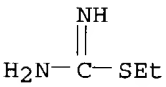


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 17 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 200134-81-2 REGISTRY
CN Carbamimidothioic acid, ethyl ester, mono(4-methylbenzenesulfonate) (9CI) (CA INDEX NAME)
MF C7 H8 O3 S . C3 H8 N2 S
SR CAS Client Services
LC STN Files: CHEMCATS

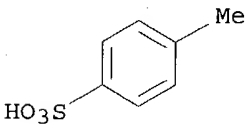
CM 1

CRN 2986-20-1
CMF C3 H8 N2 S



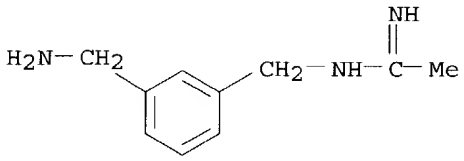
CM 2

CRN 104-15-4
CMF C7 H8 O3 S



L160 ANSWER 18 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **180001-34-7** REGISTRY
CN Ethanimidamide, N-[[3-(aminomethyl)phenyl]methyl]- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1400W
CN W 1400
FS 3D CONCORD
MF C10 H15 N3

CI COM
SR CA
LC STN Files: BIOSIS, CA, CAPLUS, CHEMCATS, CSCHEM, SYNTHLINE, TOXCENTER, USPATFULL

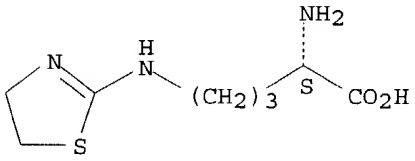


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

42 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
42 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 19 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 179116-20-2 REGISTRY
CN L-Ornithine, N5-(4,5-dihydro-2-thiazolyl)-, monohydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H15 N3 O2 S . Cl H
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER
CRN (163490-54-8)

Absolute stereochemistry.

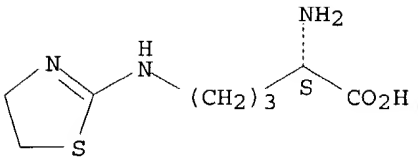


● HCl

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 20 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 177532-74-0 REGISTRY
CN L-Ornithine, N5-(4,5-dihydro-2-thiazolyl)-, dihydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H15 N3 O2 S . 2 Cl H
SR CA
LC STN Files: CA, CAPLUS, USPATFULL
CRN (163490-54-8)

Absolute stereochemistry.



●2 HCl

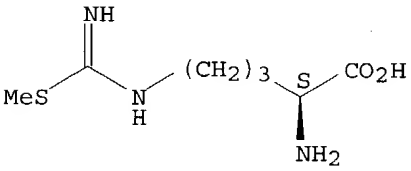
2 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 21 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 174063-92-4 REGISTRY
CN L-Ornithine, N5-[imino(methylthio)methyl]-, monoacetate (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H15 N3 O2 S . C2 H4 O2
SR CAS Client Services
LC STN Files: CHEMCATS

CM 1

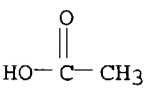
CRN 156719-41-4
CMF C7 H15 N3 O2 S

Absolute stereochemistry. Rotation (+).



CM 2

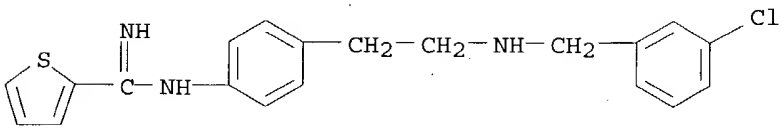
CRN 64-19-7
CMF C2 H4 O2



L160 ANSWER 22 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 168895-10-1 REGISTRY
CN 2-Thiophenecarboximidamide, N-[4-[2-[[(3-chlorophenyl)methyl]amino]ethyl]phenyl]-, ethanedioate (1:2) (9CI) (CA INDEX NAME)
MF C20 H20 Cl N3 S . 2 C2 H2 O4
SR CA
LC STN Files: CA, CAPLUS, USPATFULL

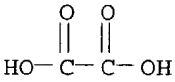
CM 1

CRN 168895-09-8
CMF C20 H20 Cl N3 S



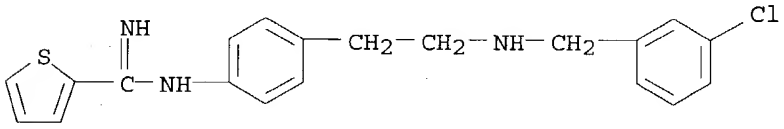
CM 2

CRN 144-62-7
CMF C2 H2 O4



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 23 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **168895-09-8** REGISTRY
CN 2-Thiophenecarboximidamide, N-[4-[2-[[(3-chlorophenyl)methyl]amino]ethyl]phenyl]- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN AR-R 17477
FS 3D CONCORD
MF C20 H20 Cl N3 S
CI COM
SR CA
LC STN Files: BIOSIS, CA, CAPLUS, TOXCENTER, USPATFULL



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

9 REFERENCES IN FILE CA (1907 TO DATE)
9 REFERENCES IN FILE CAPLUS (1907 TO DATE)

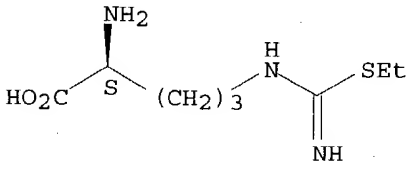
L160 ANSWER 24 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 163490-83-3 REGISTRY
CN L-Ornithine, N5-[(ethylthio)iminomethyl]-, bis(trifluoroacetate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H17 N3 O2 S . 2 C2 H F3 O2

SR CA
LC STN Files: CA, CAPLUS, USPAT2, USPATFULL

CM 1

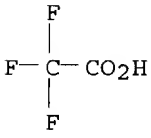
CRN 158875-72-0
CMF C8 H17 N3 O2 S

Absolute stereochemistry.



CM 2

CRN 76-05-1
CMF C2 H F3 O2



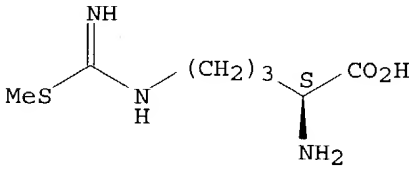
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 25 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 163490-82-2 REGISTRY
CN L-Ornithine, N5-[imino(methylthio)methyl]-, mono(trifluoroacetate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H15 N3 O2 S . C2 H F3 O2
SR CA
LC STN Files: CA, CAPLUS, USPAT2, USPATFULL

CM 1

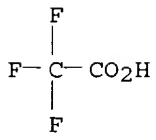
CRN 156719-41-4
CMF C7 H15 N3 O2 S

Absolute stereochemistry. Rotation (+).



CM 2

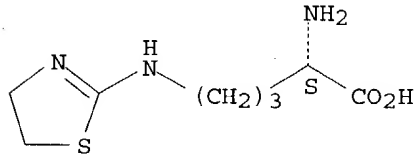
CRN 76-05-1
CMF C2 H F3 O2



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 26 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **163490-54-8** REGISTRY
CN L-Ornithine, N5-(4,5-dihydro-2-thiazolyl)- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
DR 449181-59-3
MF C8 H15 N3 O2 S
CI COM
SR CA
LC STN Files: CA, CAPLUS, USPAT2, USPATFULL

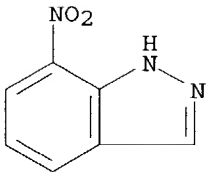
Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 27 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 161467-34-1 REGISTRY
CN 1H-Indazole, 7-nitro-, sodium salt (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 7-Nitroindazole sodium salt
MF C7 H5 N3 O2 . Na
SR CA
LC STN Files: BIOSIS, CA, CAPLUS, CHEMCATS
CRN (2942-42-9)

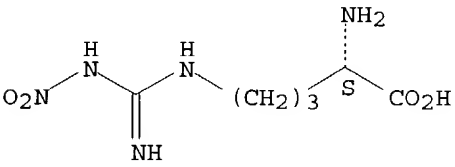


● Na

3 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 28 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 161294-82-2 REGISTRY
CN L-Ornithine, N5-[imino(nitroamino)methyl]-, monohydrochloride, monohydrate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN L-Nitroarginine monohydrochloride monohydrate
FS STEREOSEARCH
MF C6 H13 N5 O4 . Cl H . H2 O
SR CA
LC STN Files: CA, CAPLUS
CRN (2149-70-4)

Absolute stereochemistry.



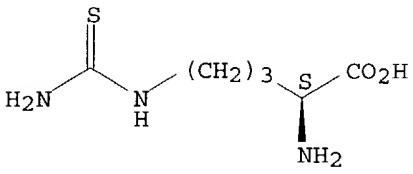
● HCl

● H_2O

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 29 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 160901-63-3 REGISTRY
CN L-Ornithine, N5-(aminothioxomethyl)-, monohydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C6 H13 N3 O2 S . Cl H
SR CAS Client Services
LC STN Files: CSCHEM
CRN (156719-37-8)

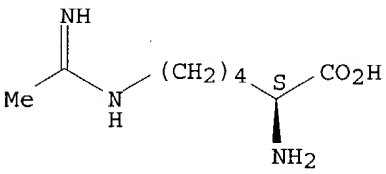
Absolute stereochemistry.



● HCl

L160 ANSWER 30 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 159190-45-1 REGISTRY
CN L-Lysine, N6-(1-iminoethyl)-, dihydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H17 N3 O2 . 2 Cl H
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, CSCHEM, TOXCENTER
CRN (53774-63-3)

Absolute stereochemistry.

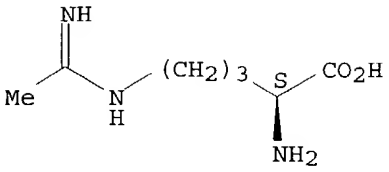


●2 HCl

11 REFERENCES IN FILE CA (1907 TO DATE)
11 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 31 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 159190-44-0 REGISTRY
CN L-Ornithine, N5-(1-iminoethyl)-, dihydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H15 N3 O2 . 2 Cl H
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER
CRN (36889-13-1)

Absolute stereochemistry.

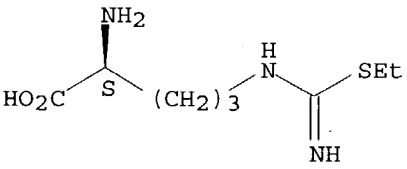


●2 HCl

3 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 32 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **158875-72-0** REGISTRY
CN L-Ornithine, N5-[(ethylthio)iminomethyl]- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN S-Ethyl-L-thiocitrulline
FS STEREOSEARCH
MF C8 H17 N3 O2 S
CI COM
SR CA
LC STN Files: BIOSIS, CA, CAPLUS, TOXCENTER, USPAT2, USPATFULL

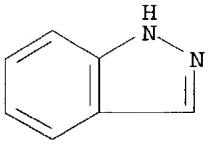
Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

11 REFERENCES IN FILE CA (1907 TO DATE)
11 REFERENCES IN FILE CAPLUS (1907 TO DATE)

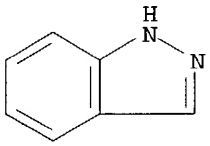
L160 ANSWER 33 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 158553-83-4 REGISTRY
CN 1H-Indazole, dihydrate (9CI) (CA INDEX NAME)
MF C7 H6 N2 . 2 H2 O
SR CA
LC STN Files: CA, CAPLUS
CRN (271-44-3)



● 2 H_2O

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

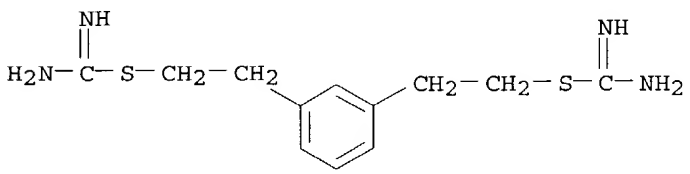
L160 ANSWER 34 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 158553-81-2 REGISTRY
CN 1H-Indazole, monohydrate (9CI) (CA INDEX NAME)
MF C7 H6 N2 . H2 O
SR CA
LC STN Files: CA, CAPLUS
CRN (271-44-3)



● H_2O

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 35 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **157254-42-7** REGISTRY
CN Carbamimidothioic acid, 1,3-phenylenedi-2,1-ethanediyl ester (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1,3-PBIT
FS 3D CONCORD
MF C12 H18 N4 S2
CI COM
SR CA
LC STN Files: BIOSIS, CA, CAPLUS, TOXCENTER, USPATFULL

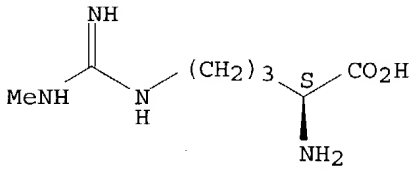


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

10 REFERENCES IN FILE CA (1907 TO DATE)
10 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 36 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 156730-43-7 REGISTRY
CN L-Ornithine, N5-[imino(methylamino)methyl]-, dihydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H16 N4 O2 . 2 Cl H
SR CA
CRN (17035-90-4)

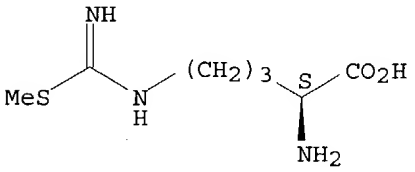
Absolute stereochemistry.



●2 HCl

L160 ANSWER 37 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **156719-41-4** REGISTRY
CN L-Ornithine, N5-[imino(methylthio)methyl]- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN S-Methyl-L-thiocitrulline
CN S-Methylthiocitrulline
CN S-MTC
FS STEREOSEARCH
MF C7 H15 N3 O2 S
CI COM
SR CA
LC STN Files: BIOSIS, CA, CANCERLIT, CAPLUS, CEN, CHEMCATS, MEDLINE, TOXCENTER, USPAT2, USPATFULL

Absolute stereochemistry. Rotation (+).



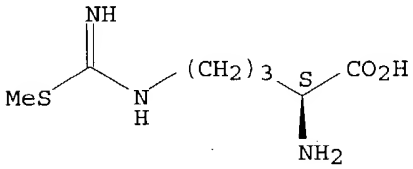
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

40 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

40 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 38 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 156719-39-0 REGISTRY
CN L-Ornithine, N5-[imino(methylthio)methyl]-, monohydrochloride (9CI) (CA INDEX NAME)
OTHER NAMES:
CN S-Methylthiocitrulline hydrochloride
FS STEREOSEARCH
MF C7 H15 N3 O2 S . Cl H
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, CSCHEM, TOXCENTER
CRN (156719-41-4)

Absolute stereochemistry. Rotation (+).

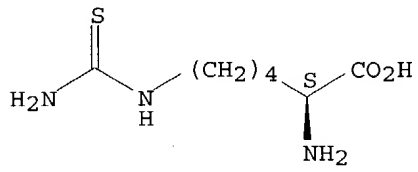


● HCl

4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 39 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **156719-38-9** REGISTRY
CN L-Lysine, N6-(aminothioxomethyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN L-Homothiocitrulline
FS STEREOSEARCH
MF C7 H15 N3 O2 S
CI COM
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



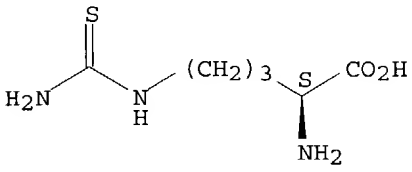
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 40 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **156719-37-8** REGISTRY

CN L-Ornithine, N5-(aminothioxomethyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN L-TC
CN L-Thiocitrulline
FS STEREOSEARCH
MF C6 H13 N3 O2 S
CI COM
SR CA
LC STN Files: BIOSIS, CA, CAPLUS, CHEMCATS, CSCHEM, TOXCENTER, USPATFULL

Absolute stereochemistry.

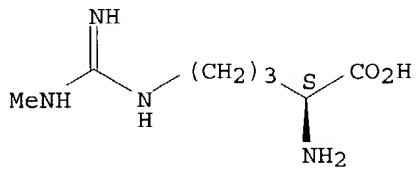


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

41 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
41 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 41 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 156706-47-7 REGISTRY
CN L-Ornithine, N5-[imino(methylamino)methyl]-, monohydrochloride (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 546C88
CN NG-Monomethyl-L-arginine hydrochloride
FS STEREOSEARCH
MF C7 H16 N4 O2 . Cl H
SR CA
LC STN Files: ADISINSIGHT, BIOTECHNO, CA, CAPLUS, CASREACT, CHEMCATS, CIN, EMBASE, IMSDRUGNEWS, IMSPATENTS, IMSRESEARCH, IPA, SYNTHLINE, TOXCENTER, USPATFULL
CRN (17035-90-4)

Absolute stereochemistry.



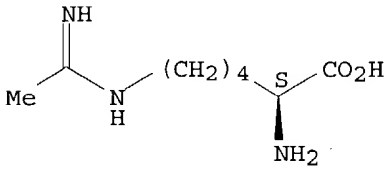
● HCl

7 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 42 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN

RN 150403-89-7 REGISTRY
CN L-Lysine, N6-(1-iminoethyl)-, monohydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H17 N3 O2 . Cl H
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, CSCHEM, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
CRN (53774-63-3)

Absolute stereochemistry.

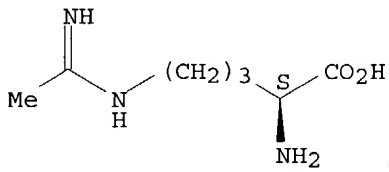


● HCl

8 REFERENCES IN FILE CA (1907 TO DATE)
8 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 43 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 150403-88-6 REGISTRY
CN L-Ornithine, N5-(1-iminoethyl)-, monohydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H15 N3 O2 . Cl H
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, CSCHEM, TOXCENTER, USPAT2, USPATFULL
CRN (36889-13-1)

Absolute stereochemistry.



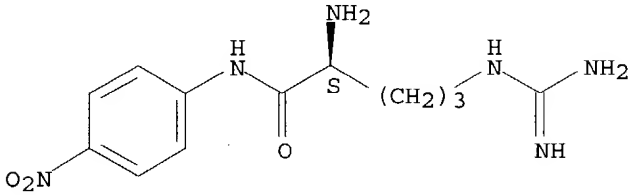
● HCl

7 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 44 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **140845-10-9** REGISTRY
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, dihydrofluoride, (S)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Arginine p-nitroanilide dihydrofluoride
FS STEREOSEARCH
MF C12 H18 N6 O3 . 2 F H

SR CA
LC STN Files: CA, CAPLUS
CRN (6154-84-3)

Absolute stereochemistry.

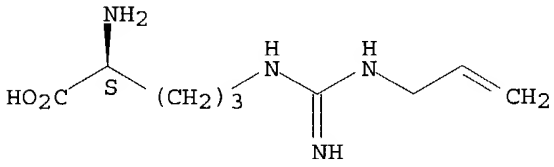


●2 HF

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 45 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **139461-37-3** REGISTRY
CN L-Ornithine, N5-[imino(2-propenylamino)methyl]- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C9 H18 N4 O2
CI COM
SR CA
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMINFORMRX, MEDLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.

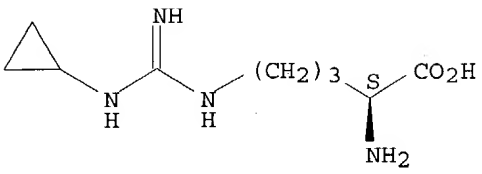


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

13 REFERENCES IN FILE CA (1907 TO DATE)
13 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 46 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **139299-32-4** REGISTRY
CN L-Ornithine, N5-[(cyclopropylamino)iminomethyl]- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C9 H18 N4 O2
CI COM
SR CA
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, MEDLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

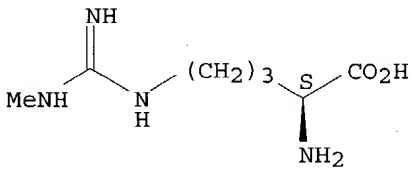
8 REFERENCES IN FILE CA (1907 TO DATE)
8 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 47 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 133587-00-5 REGISTRY
CN L-Ornithine, N5-[imino(methylamino)methyl]-, acetate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN NG-monomethyl-L-arginine acetate
FS STEREOSEARCH
DR 139953-34-7
MF C7 H16 N4 O2 . x C2 H4 O2
SR CA
LC STN Files: CA, CAPLUS, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

CM 1

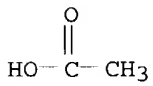
CRN 17035-90-4
CMF C7 H16 N4 O2

Absolute stereochemistry.



CM 2

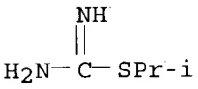
CRN 64-19-7
CMF C2 H4 O2



13 REFERENCES IN FILE CA (1907 TO DATE)
13 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 48 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN

RN 127118-67-6 REGISTRY
CN Carbamimidothioic acid, 1-methylethyl ester, monohydriodide (9CI) (CA INDEX NAME)
MF C4 H10 N2 S . H I
SR CA
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT
(*File contains numerically searchable property data)
CRN (6913-17-3)

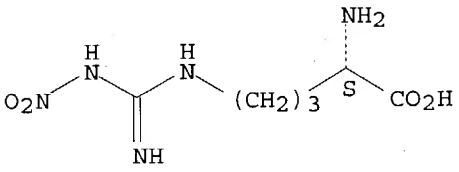


● HI

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 49 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 115699-60-0 REGISTRY
CN L-Ornithine, N5-[imino(nitroamino)methyl]-, monopotassium salt (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C6 H13 N5 O4 . K
SR CA
LC STN Files: CA, CAPLUS
CRN (2149-70-4)

Absolute stereochemistry.

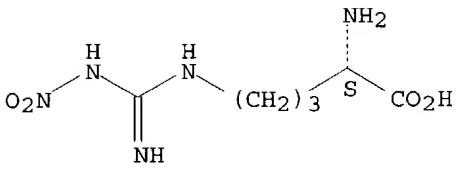


● K

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 50 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 115699-59-7 REGISTRY
CN L-Ornithine, N5-[imino(nitroamino)methyl]-, monosodium salt (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C6 H13 N5 O4 . Na
SR CA
LC STN Files: CA, CAPLUS
CRN (2149-70-4)

Absolute stereochemistry.



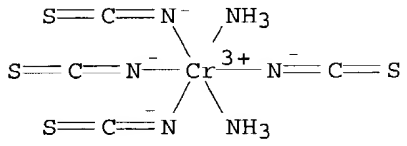
● Na

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 51 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 114698-01-0 REGISTRY
CN Pseudourea, 2-methyl-2-thio-, reineckate (6CI) (CA INDEX NAME)
MF C4 H6 Cr N6 S4 . C2 H6 N2 S . H
SR CAOLD
LC STN Files: CA, CAOLD, CAPLUS

CM 1

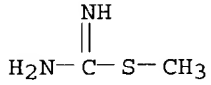
CRN 16925-04-5 (16248-93-4)
CMF C4 H6 Cr N6 S4 . H
CCI CCS



● H^+ .

CM 2

CRN 2986-19-8
CMF C2 H6 N2 S

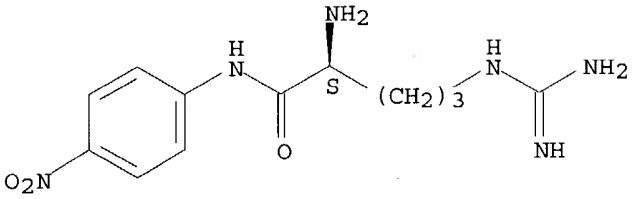


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 52 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 113277-28-4 REGISTRY

CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, hydrobromide, (S)- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C12 H18 N6 O3 . x Br H
SR CA
LC STN Files: CA, CAPLUS
CRN (6154-84-3)

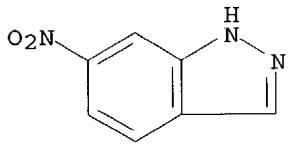
Absolute stereochemistry.



● x HBr

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

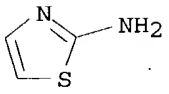
L160 ANSWER 53 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 104436-77-3 REGISTRY
CN 1H-Indazole, 6-nitro-, sodium salt (9CI) (CA INDEX NAME)
DR 126874-71-3
MF C7 H5 N3 O2 . Na
SR CA
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, USPATFULL
(*File contains numerically searchable property data)
CRN (7597-18-4)



● Na

4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 54 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 102862-49-7 REGISTRY
CN 2-Thiazolamine, monohydrobromide (9CI) (CA INDEX NAME)
MF C3 H4 N2 S . Br H
SR CA
LC STN Files: CA, CAPLUS
CRN (96-50-4)



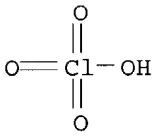
● HBr

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 55 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 95675-87-9 REGISTRY
CN Carbamimidothioic acid, ethyl ester, monoperchlorate (9CI) (CA INDEX NAME)
MF C3 H8 N2 S . Cl H O4
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT
(*File contains numerically searchable property data)

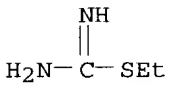
CM 1

CRN 7601-90-3
CMF Cl H O4



CM 2

CRN 2986-20-1
CMF C3 H8 N2 S

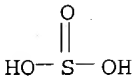


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 56 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 93857-62-6 REGISTRY
CN 2-Thiazolamine, sulfite (1:1) (9CI) (CA INDEX NAME)
MF C3 H4 N2 S . H2 O3 S
SR European Union (EU)
LC STN Files: CHEMLIST
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

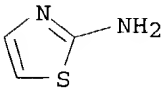
CM 1

CRN 7782-99-2
CMF H2 O3 S



CM 2

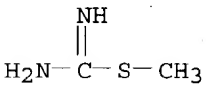
CRN 96-50-4
CMF C3 H4 N2 S



L160 ANSWER 57 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 92765-38-3 REGISTRY
CN Pseudourea, 2-methyl-2-thio-, benzoate (7CI) (CA INDEX NAME)
MF C7 H6 O2 . C2 H6 N2 S
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS
(*File contains numerically searchable property data)

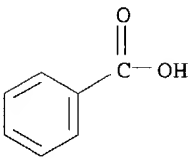
CM 1

CRN 2986-19-8
CMF C2 H6 N2 S



CM 2

CRN 65-85-0
CMF C7 H6 O2

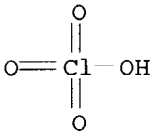


2 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 58 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 92303-85-0 REGISTRY
CN 1H-Indazole, perchlorate (7CI) (CA INDEX NAME)
MF C7 H6 N2 . Cl H O4
LC STN Files: CA, CAOLD, CAPLUS

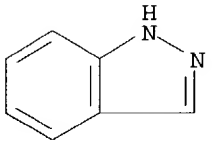
CM 1

CRN 7601-90-3
CMF Cl H O4



CM 2

CRN 271-44-3
CMF C7 H6 N2

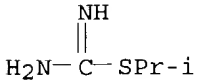


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 59 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 90973-53-8 REGISTRY
CN Pseudourea, 2-isopropyl-2-thio-, picrate (7CI) (CA INDEX NAME)
MF C6 H3 N3 O7 . C4 H10 N2 S
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CHEMCATS
(*File contains numerically searchable property data)

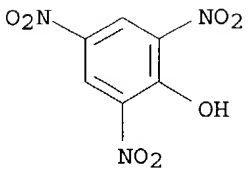
CM 1

CRN 6913-17-3
CMF C4 H10 N2 S



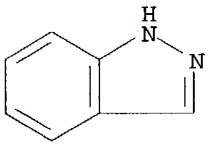
CM 2

CRN 88-89-1
CMF C6 H3 N3 O7



7 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

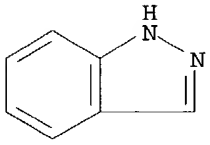
L160 ANSWER 60 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 83482-89-7 REGISTRY
CN 1H-Indazole, silver(1+) salt (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Indazole silver salt
MF C7 H6 N2 . Ag
LC STN Files: BEILSTEIN*, CA, CAPLUS, GMELIN*
(*File contains numerically searchable property data)
CRN (271-44-3)



● Ag(I)

2 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 61 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 81252-84-8 REGISTRY
CN 1H-Indazole, monohydrobromide (9CI) (CA INDEX NAME)
MF C7 H6 N2 . Br H
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT
(*File contains numerically searchable property data)
CRN (271-44-3)



● HBr

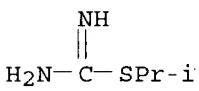
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

2 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 62 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 77912-98-2 REGISTRY
CN Carbamimidothioic acid, 1-methylethyl ester, conjugate monoacid, salt with 2,2'-(2,5-cyclohexadiene-1,4-diylidene)bis[propanedinitrile] (1:1) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Propanedinitrile, 2,2'-(2,5-cyclohexadiene-1,4-diylidene)bis-, radical ion(1-), salt with 1-methylethyl carbamimidothioate conjugate monoacid (1:1) (9CI)
MF C12 H4 N4 . C4 H10 N2 S . H
LC STN Files: CA, CAPLUS

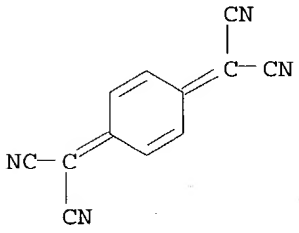
CM 1

CRN 77912-97-1 (6913-17-3)
CMF C4 H10 N2 S . H



CM 2

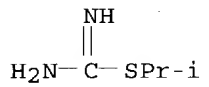
CRN 34507-61-4
CMF C12 H4 N4
CCI RIS



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 63 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 77912-97-1 REGISTRY
CN Carbamimidothioic acid, 1-methylethyl ester, conjugate monoacid (9CI) (CA INDEX NAME)

MF C4 H10 N2 S . H
CI COM
LC STN Files: CA, CAPLUS
CRN (6913-17-3)

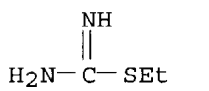


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 64 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 77912-94-8 REGISTRY
CN Carbamimidothioic acid, ethyl ester, conjugate monoacid, salt with 2,2'-(2,5-cyclohexadiene-1,4-diylidene)bis[propanedinitrile] (1:1) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Propanedinitrile, 2,2'-(2,5-cyclohexadiene-1,4-diylidene)bis-, radical ion(1-), salt with ethyl carbamimidothioate conjugate monoacid (1:1) (9CI)
MF C12 H4 N4 . C3 H8 N2 S . H
LC STN Files: CA, CAPLUS

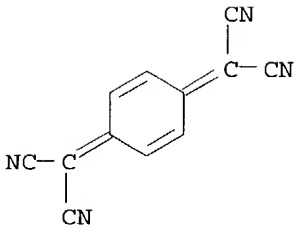
CM 1

CRN 77912-93-7 (2986-20-1)
CMF C3 H8 N2 S . H



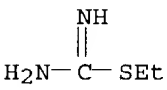
CM 2

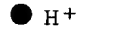
CRN 34507-61-4
CMF C12 H4 N4
CCI RIS



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 65 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 77912-93-7 REGISTRY
CN Carbamimidothioic acid, ethyl ester, conjugate monoacid (9CI) (CA INDEX NAME)
MF C3 H8 N2 S . H
CI COM
LC STN Files: CA, CAPLUS
CRN (2986-20-1)



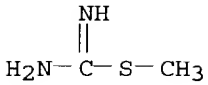


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 66 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 77912-92-6 REGISTRY
CN Carbamimidothioic acid, methyl ester, conjugate monoacid, salt with 2,2'-(2,5-cyclohexadiene-1,4-diylidene)bis[propanedinitrile] (1:1) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Propanedinitrile, 2,2'-(2,5-cyclohexadiene-1,4-diylidene)bis-, radical ion(1-), salt with methyl carbamimidothioate conjugate monoacid (1:1) (9CI)
MF C12 H4 N4 . C2 H6 N2 S . H
CI COM
LC STN Files: CA, CAPLUS

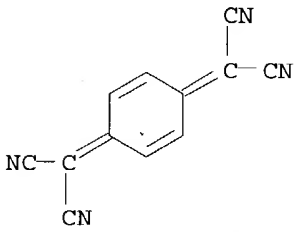
CM 1

CRN 54496-46-7 (2986-19-8)
CMF C2 H6 N2 S . H



CM 2

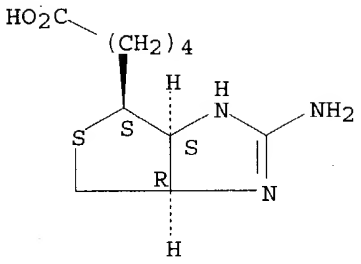
CRN 34507-61-4
CMF C12 H4 N4
CCI RIS



4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 67 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 76985-52-9 REGISTRY
CN 1H-Thieno[3,4-d]imidazole-4-pentanoic acid, 2-amino-3a,4,6,6a-tetrahydro-, monohydrobromide, [3aS-(3aα,4β,6aα)]- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Iminobiotin hydrobromide
FS STEREOSEARCH
MF C10 H17 N3 O2 S . Br H
LC STN Files: BEILSTEIN*, CA, CAPLUS
(*File contains numerically searchable property data)
CRN (13395-35-2)

Absolute stereochemistry.



● HBr

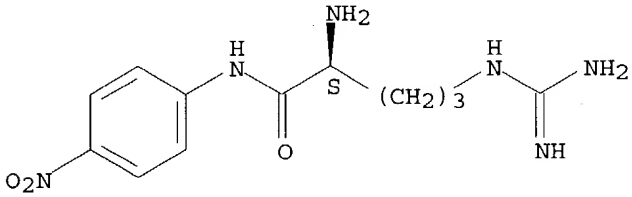
2 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 68 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 75736-19-5 REGISTRY
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, (S)-, monoacetate (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C12 H18 N6 O3 . C2 H4 O2
LC STN Files: CA, CAPLUS, USPATFULL

CM 1

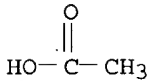
CRN 6154-84-3
CMF C12 H18 N6 O3

Absolute stereochemistry.



CM 2

CRN 64-19-7
CMF C2 H4 O2

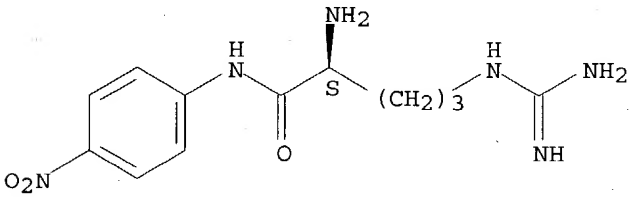


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 69 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN

RN 75613-02-4 REGISTRY
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, monohydrochloride, (2S)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, monohydrochloride, (S)-
FS STEREOSEARCH
MF C12 H18 N6 O3 . Cl H
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT
(*File contains numerically searchable property data)
CRN (6154-84-3)

Absolute stereochemistry.



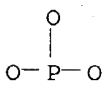
● HCl

4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 70 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 72333-43-8 REGISTRY
CN Carbamimidothioic acid, methyl ester, phosphonate (1:1) (9CI) (CA INDEX NAME)
MF C2 H6 N2 S . H3 O3 P
LC STN Files: CA, CAPLUS, USPATFULL

CM 1

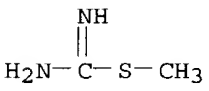
CRN 13598-36-2
CMF H3 O3 P



*** FRAGMENT DIAGRAM IS INCOMPLETE ***

CM 2

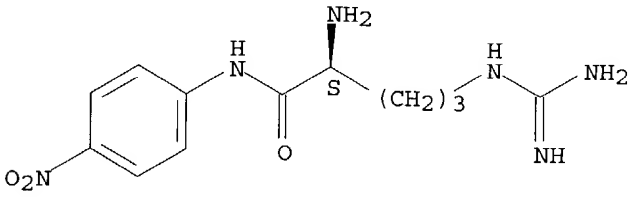
CRN 2986-19-8
CMF C2 H6 N2 S



3 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 71 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 72244-04-3 REGISTRY
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, monohydrobromide, (S)- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C12 H18 N6 O3 . Br H
LC STN Files: CA, CAPLUS, CASREACT, TOXCENTER, USPATFULL
CRN (6154-84-3)

Absolute stereochemistry.

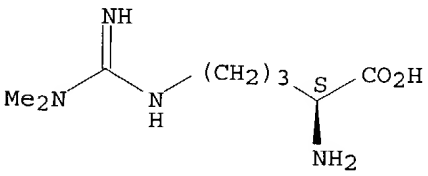


● HBr

5 REFERENCES IN FILE CA (1907 TO DATE)
5 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 72 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 65005-57-4 REGISTRY
CN L-Ornithine, N5-[(dimethylamino)iminomethyl]-, monohydrochloride (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C8 H18 N4 O2 . Cl H
LC STN Files: CA, CAPLUS, CHEMCATS, CSCHEM
CRN (30315-93-6)

Absolute stereochemistry.

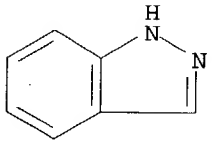


● HCl

6 REFERENCES IN FILE CA (1907 TO DATE)
6 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 73 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 63725-55-3 REGISTRY
CN 1H-Indazole, monohydrochloride (9CI) (CA INDEX NAME)

MF C7 H6 N2 . Cl H
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, TOXCENTER
(*File contains numerically searchable property data)
CRN (271-44-3)



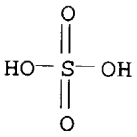
● HCl

5 REFERENCES IN FILE CA (1907 TO DATE)
5 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 74 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 63589-20-8 REGISTRY
CN 2-Thiazolamine, sulfate (1:1) (9CI) (CA INDEX NAME)
MF C3 H4 N2 S . H2 O4 S
LC STN Files: CA, CAPLUS, CHEMLIST, USPATFULL
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

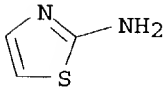
CM 1

CRN 7664-93-9
CMF H2 O4 S



CM 2

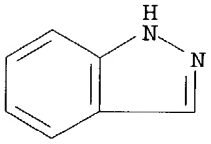
CRN 96-50-4
CMF C3 H4 N2 S



2 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 75 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 63566-71-2 REGISTRY
CN 1H-Indazole, copper(2+) salt (9CI) (CA INDEX NAME)

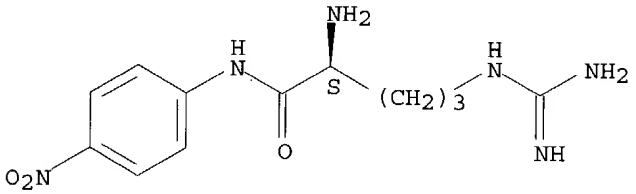
MF C7 H6 N2 . 1/2 Cu
CI COM
CRN (271-44-3)



●1/2 Cu(II)

L160 ANSWER 76 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 61876-73-1 REGISTRY
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, dihydrobromide, (S)- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C12 H18 N6 O3 . 2 Br H
LC STN Files: CA, CAPLUS, IFICDB, IFIPAT, IFIUDB, USPATFULL
CRN (6154-84-3)

Absolute stereochemistry.



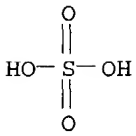
●2 HBr

10 REFERENCES IN FILE CA (1907 TO DATE)
10 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 77 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 61169-63-9 REGISTRY
CN 2-Thiazolamine, sulfate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2-Aminothiazole sulfate
MF C3 H4 N2 S . x H2 O4 S
LC STN Files: CA, CAPLUS, CHEMCATS, USPATFULL

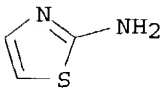
CM 1

CRN 7664-93-9
CMF H2 O4 S



CM 2

CRN 96-50-4
CMF C3 H4 N2 S

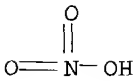


4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 78 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 57530-25-3 REGISTRY
CN 2-Thiazolamine, mononitrate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2-Aminothiazole nitrate
MF C3 H4 N2 S . H N O3
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAPLUS, CHEMLIST, CIN, CSCHEM
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

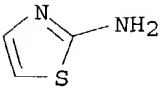
CM 1

CRN 7697-37-2
CMF H N O3



CM 2

CRN 96-50-4
CMF C3 H4 N2 S

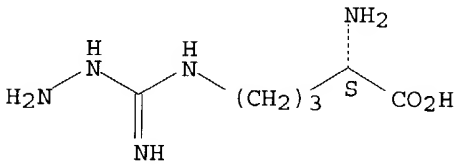


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 79 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **57444-72-1** REGISTRY
CN L-Ornithine, N5-(hydrazinoiminomethyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN NG-Amino-L-arginine
FS STEREOSEARCH
MF C6 H15 N5 O2
CI COM
LC STN Files: CA, CANCERLIT, CAPLUS, MEDLINE, TOXCENTER, USPAT2, USPATFULL

Absolute stereochemistry.



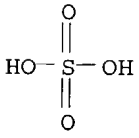
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

47 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
47 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 80 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 57200-31-4 REGISTRY
CN Carbamimidothioic acid, 1-methylethyl ester, sulfate (1:1) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN S-Isopropylisothiourea sulfate
MF C4 H10 N2 S . H2 O4 S
LC STN Files: BEILSTEIN*, CA, CAPLUS, CHEMCATS, IFICDB, IFIPAT, IFIUDB
(*File contains numerically searchable property data)

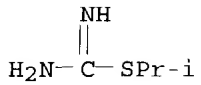
CM 1

CRN 7664-93-9
CMF H2 O4 S



CM 2

CRN 6913-17-3
CMF C4 H10 N2 S

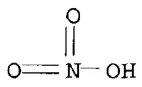


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 81 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 56698-48-7 REGISTRY
CN Carbamimidothioic acid, methyl ester, mononitrate (9CI) (CA INDEX NAME)
MF C2 H6 N2 S . H N O3
LC STN Files: BEILSTEIN*, CA, CAPLUS, IFICDB, IFIPAT, IFIUDB
(*File contains numerically searchable property data)

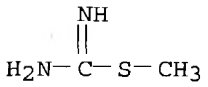
CM 1

CRN 7697-37-2
CMF H N O3



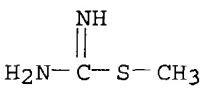
CM 2

CRN 2986-19-8
CMF C2 H6 N2 S



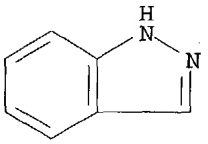
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 82 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 54496-46-7 REGISTRY
CN Carbamimidothioic acid, methyl ester, conjugate monoacid (9CI) (CA INDEX NAME)
MF C2 H6 N2 S . H
CI COM
LC STN Files: CA, CAPLUS, CASREACT
CRN (2986-19-8)



7 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 83 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 53857-56-0 REGISTRY
CN 1H-Indazole, conjugate monoacid (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Indazole conjugate monoacid
MF C7 H6 N2 . H
LC STN Files: CA, CAPLUS
CRN (271-44-3)

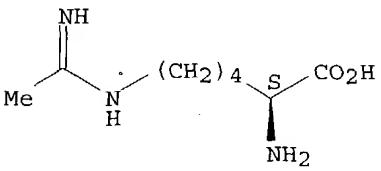


● H^+

6 REFERENCES IN FILE CA (1907 TO DATE)
6 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 84 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **53774-63-3** REGISTRY
CN L-Lysine, N6-(1-iminoethyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN ε-Acetimidyllysine
CN L-NIL
FS STEREOSEARCH
MF C8 H17 N3 O2
CI COM
LC STN Files: ANABSTR, BIOSIS, BIOTECHNO, CA, CAPLUS, CHEMCATS, EMBASE, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

75 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
75 REFERENCES IN FILE CAPLUS (1907 TO DATE)

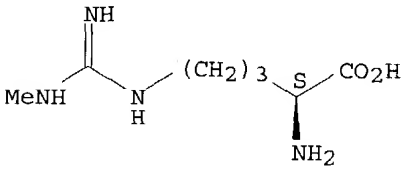
L160 ANSWER 85 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 53308-83-1 REGISTRY

CN L-Ornithine, N5-[imino(methylamino)methyl]-, monoacetate (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C7 H16 N4 O2 . C2 H4 O2
LC STN Files: ADISINSIGHT, BIOSIS, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, EMBASE, MEDLINE, SYNTHLINE, TOXCENTER

CM 1

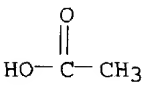
CRN 17035-90-4
CMF C7 H16 N4 O2

Absolute stereochemistry.



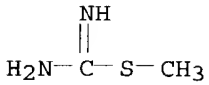
CM 2

CRN 64-19-7
CMF C2 H4 O2



11 REFERENCES IN FILE CA (1907 TO DATE)
11 REFERENCES IN FILE CAPLUS (1907 TO DATE)

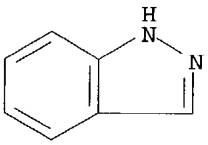
L160 ANSWER 86 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 53114-57-1 REGISTRY
CN Carbamimidothioic acid, methyl ester, monohydrochloride (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Methyl isothiuronium chloride
CN S-Methylisothiourea chloride
CN S-Methylisothiourea hydrochloride
CN S-Methylisothiuronium chloride
DR 16516-49-7, 83354-16-9, 38750-51-5
MF C2 H6 N2 S . Cl H
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMINFORMRX, CHEMLIST, CSCHEM, IFICDB, IFIPAT, IFIUDB, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (2986-19-8)



● HCl

35 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
35 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 87 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 41253-33-2 REGISTRY
CN 1H-Indazole, sodium salt (9CI) (CA INDEX NAME)
MF C7 H6 N2 . Na
LC STN Files: CA, CAPLUS, TOXCENTER
CRN (271-44-3)

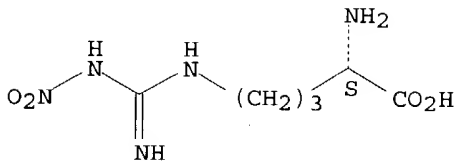


● Na

6 REFERENCES IN FILE CA (1907 TO DATE)
6 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 88 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 40911-12-4 REGISTRY
CN L-Ornithine, N5-[imino(nitroamino)methyl]-, monohydrochloride (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Nω-Nitro-L-arginine hydrochloride
CN Ng-Nitroarginine hydrochloride
CN Nitroargine monohydrochloride
FS STEREOSEARCH
DR 54046-21-8, 115134-28-6
MF C6 H13 N5 O4 . Cl H
LC STN Files: CA, CAPLUS, CASREACT
CRN (2149-70-4)

Absolute stereochemistry.



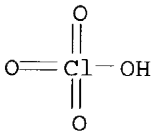
● HCl

7 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 89 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 40253-83-6 REGISTRY
CN 2-Thiazolamine, monoperchlorate (9CI) (CA INDEX NAME)
MF C3 H4 N2 S . Cl H O4
LC STN Files: BEILSTEIN*, CA, CAPLUS
(*File contains numerically searchable property data)

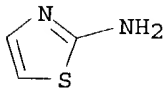
CM 1

CRN 7601-90-3
CMF Cl H O4



CM 2

CRN 96-50-4
CMF C3 H4 N2 S

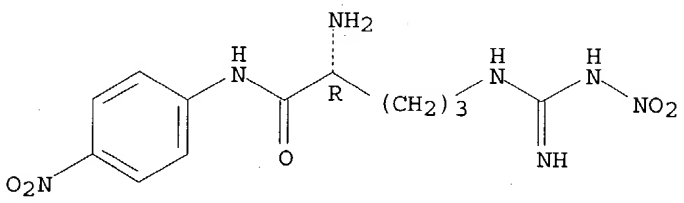


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 90 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **40127-15-9** REGISTRY
CN Pentanamide, 2-amino-5-[[imino(nitroamino)methyl]amino]-N-(4-nitrophenyl)-, monohydrobromide, (R)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Nω-Nitro-D-arginine p-nitroanilide hydrobromide
FS STEREOSEARCH
MF C12 H17 N7 O5 . Br H
LC STN Files: BEILSTEIN*, CA, CAPLUS

(*File contains numerically searchable property data)

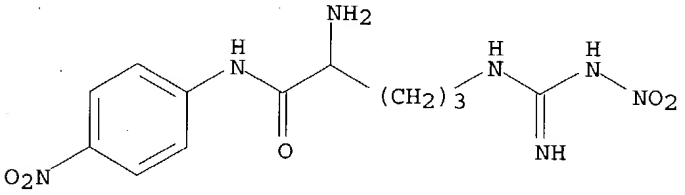
Absolute stereochemistry.



● HBr

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 91 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **40127-14-8** REGISTRY
CN Pentanamide, 2-amino-5-[[imino(nitroamino)methyl]amino]-N-(4-nitrophenyl)-, monohydrobromide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pentanamide, 2-amino-5-[[imino(nitroamino)methyl]amino]-N-(4-nitrophenyl)-, monohydrobromide, (±)-
OTHER NAMES:
CN Nω-Nitro-DL-arginine p-nitroanilide hydrobromide
MF C12 H17 N7 O5 . Br H
LC STN Files: BEILSTEIN*, CA, CAPLUS, CHEMCATS
(*File contains numerically searchable property data)



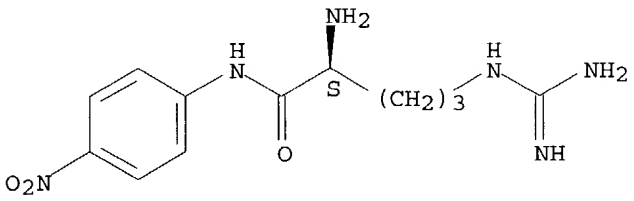
● HBr

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 92 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 40127-11-5 REGISTRY
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, dihydrochloride, (2S)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, dihydrochloride, (S)-
OTHER NAMES:
CN L-Arginine p-nitroanilide dihydrochloride
FS STEREOSEARCH

MF C12 H18 N6 O3 . 2 Cl H
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, MSDS-OHS, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
CRN (6154-84-3)

Absolute stereochemistry.



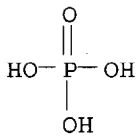
●2 HCl

26 REFERENCES IN FILE CA (1907 TO DATE)
26 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 93 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 36908-12-0 REGISTRY
CN Carbamimidothioic acid, methyl ester, phosphate (1:1) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN S-Methylthiuronium dihydrogen phosphate
MF C2 H6 N2 S . H3 O4 P
LC STN Files: BEILSTEIN*, CA, CAPLUS
(*File contains numerically searchable property data)

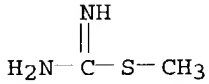
CM 1

CRN 7664-38-2
CMF H3 O4 P



CM 2

CRN 2986-19-8
CMF C2 H6 N2 S

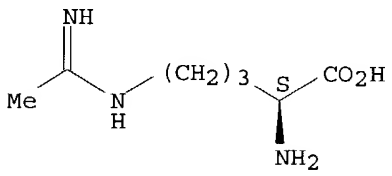


1 REFERENCES IN FILE CA (1907 TO DATE)

1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 94 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **36889-13-1** REGISTRY
CN L-Ornithine, N5-(1-iminoethyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN L-NIO
CN Nδ-(Iminoethyl)-L-ornithine
CN N5-(1-Iminoethyl)-L-ornithine
FS STEREOSEARCH
MF C7 H15 N3 O2
CI COM
LC STN Files: BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CHEMCATS, CSCHEM, EMBASE, MEDLINE, TOXCENTER, USPAT2, USPATFULL

Absolute stereochemistry.

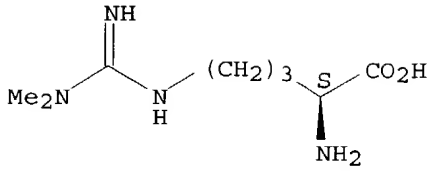


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

110 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
110 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 95 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **30315-93-6** REGISTRY
CN L-Ornithine, N5-[(dimethylamino)iminomethyl]- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ornithine, N5-(N,N-dimethylamidino)-, L- (8CI)
OTHER NAMES:
CN ADMA
CN Asymmetric dimethylarginine
CN Dimethyl-L-arginine
CN L-NG,NG-Dimethylarginine
CN Ng,Ng-Dimethylarginine
CN NG,NG-Dimethylarginine
FS STEREOSEARCH
DR 32747-09-4
MF C8 H18 N4 O2
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CIN, CSCHEM, EMBASE, MEDLINE, PROMT, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.



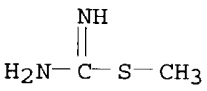
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

264 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
268 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 96 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 27950-17-0 REGISTRY
CN Pseudourea, 2-methyl-2-thio-, monoacetate (8CI) (CA INDEX NAME)
OTHER NAMES:
CN S-Methylisothiouronium acetate
MF C2 H6 N2 S . C2 H4 O2
LC STN Files: BEILSTEIN*, CA, CAPLUS, TOXCENTER
(*File contains numerically searchable property data)

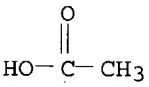
CM 1

CRN 2986-19-8
CMF C2 H6 N2 S



CM 2

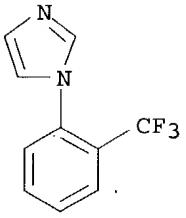
CRN 64-19-7
CMF C2 H4 O2



3 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 97 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **25371-96-4** REGISTRY
CN 1H-Imidazole, 1-[2-(trifluoromethyl)phenyl]- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Imidazole, 1-(α,α,α-trifluoro-o-tolyl)- (8CI)
OTHER NAMES:
CN 1-(2-Trifluoromethylphenyl)imidazole
FS 3D CONCORD
MF C10 H7 F3 N2

CI COM
LC STN Files: BIOSIS, CA, CANCERLIT, CAPLUS, CHEMCATS, CSCHEM, MEDLINE, TOXCENTER, USPAT2, USPATFULL



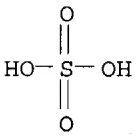
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

33 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
34 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 98 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 22722-03-8 REGISTRY
CN Carbamimidothioic acid, ethyl ester, sulfate (1:1) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-ethyl-2-thio-, sulfate (1:1) (8CI)
OTHER NAMES:
CN S-Ethylisothiourea dihydrogen sulfate
CN S-Ethylisothiourea sulfate
MF C3 H8 N2 S . H2 O4 S
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, IFICDB, IFIPAT, IFIUDB, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

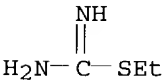
CM 1

CRN 7664-93-9
CMF H2 O4 S



CM 2

CRN 2986-20-1
CMF C3 H8 N2 S

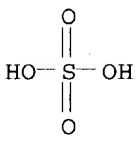


10 REFERENCES IN FILE CA (1907 TO DATE)
10 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 99 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 22410-59-9 REGISTRY
CN Carbamimidothioic acid, ethyl ester, sulfate (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-ethyl-2-thio-, sulfate (8CI)
MF C3 H8 N2 S . x H2 O4 S
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, IFICDB, IFIPAT, IFIUDB, TOXCENTER
(*File contains numerically searchable property data)

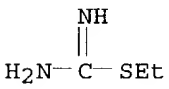
CM 1

CRN 7664-93-9
CMF H2 O4 S



CM 2

CRN 2986-20-1
CMF C3 H8 N2 S

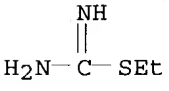


8 REFERENCES IN FILE CA (1907 TO DATE)
8 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 100 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 21704-49-4 REGISTRY
CN Pseudourea, 2-ethyl-2-thio-, citrate (3:2) (8CI) (CA INDEX NAME)
MF C6 H8 O7 . 3/2 C3 H8 N2 S
LC STN Files: CA, CAPLUS, TOXCENTER

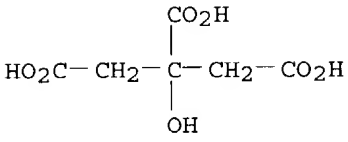
CM 1

CRN 2986-20-1
CMF C3 H8 N2 S



CM 2

CRN 77-92-9
CMF C6 H8 O7

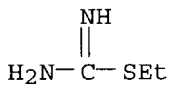


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 101 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 21704-47-2 REGISTRY
CN Pseudourea, 2-ethyl-2-thio-, monoacetate (8CI) (CA INDEX NAME)
OTHER NAMES:
CN S-Ethylisothiouronium acetate
MF C3 H8 N2 S . C2 H4 O2
LC STN Files: BEILSTEIN*, CA, CAPLUS, TOXCENTER
(*File contains numerically searchable property data)

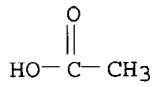
CM 1

CRN 2986-20-1
CMF C3 H8 N2 S



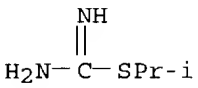
CM 2

CRN 64-19-7
CMF C2 H4 O2



4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 102 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 18939-66-7 REGISTRY
CN Pseudourea, 2-isopropyl-2-thio-, monohydrochloride (8CI) (CA INDEX NAME)
MF C4 H10 N2 S . Cl H
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, IFICDB, IFIPAT, IFIUDB
(*File contains numerically searchable property data)
CRN (6913-17-3)

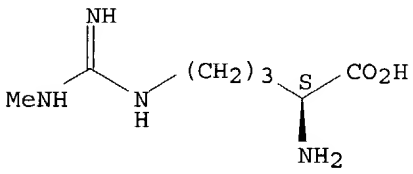


● HCl

2 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 103 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **17035-90-4** REGISTRY
CN L-Ornithine, N5-[imino(methylamino)methyl]- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ornithine, N5-(methylamidino)-, L- (8CI)
OTHER NAMES:
CN ω-N-Methylarginine
CN ω-N-Monomethylarginine
CN L-Monomethylarginine
CN L-NG-Methylarginine
CN L-NMA
CN L-NMMA
CN Methylarginine
CN N5-(Methylamidino)-L-ornithine
CN NG-Methyl-L-arginine
CN NG-methyl-L-arginine
CN NG-Methylarginine
CN NG-Monomethyl-L-arginine
CN NG-Monomethylarginine
CN Targinine
FS STEREOSEARCH
DR 42342-68-7
MF C7 H16 N4 O2
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IMSPATENTS, IMSRESEARCH, IPA, MEDLINE, PHAR, PROMT, RTECS*, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)

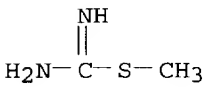
Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

859 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
862 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 104 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 16516-53-3 REGISTRY
CN Pseudourea, 2-methyl-2-thio-, hydrobromide (8CI) (CA INDEX NAME)
MF C2 H6 N2 S . x Br H
LC STN Files: BEILSTEIN*, CA, CAPLUS, IFICDB, IFIPAT, IFIUDB, TOXCENTER
(*File contains numerically searchable property data)
CRN (2986-19-8)



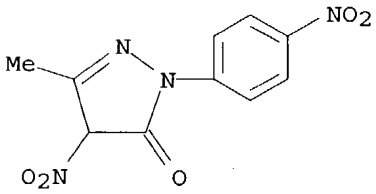
●x HBr

5 REFERENCES IN FILE CA (1907 TO DATE)
5 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 105 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 14975-86-1 REGISTRY
CN Thiazole, 2-amino-, picrolonate (8CI) (CA INDEX NAME)
MF C10 H8 N4 O5 . x C3 H4 N2 S
LC STN Files: CA, CAPLUS

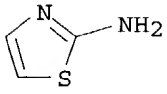
CM 1

CRN 550-74-3
CMF C10 H8 N4 O5



CM 2

CRN 96-50-4
CMF C3 H4 N2 S



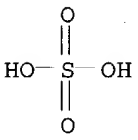
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 106 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 14527-26-5 REGISTRY
CN Carbamimidothioic acid, methyl ester, sulfate (1:1) (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:
CN Pseudourea, 2-methyl-2-thio-, sulfate (1:1) (8CI)
OTHER NAMES:
CN 2-Methylisothiouronium sulfate
CN NSC 227845
CN S-Methylisothiourea sulfate (1:1)
CN S-Methylpseudothiourea sulfate
CN S-Methylthiopseudourea hydrogen sulfate
CN S-Methylthiopseudourea sulfate (1:1)
CN S-Methylthiourea sulfate
CN S-Methylthiuronium sulfate
DR 93744-74-2
MF C2 H6 N2 S . H2 O4 S
LC STN Files: AQUIRE, BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, IFICDB, IFIPAT, IFIUDB, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

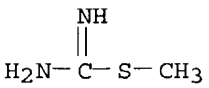
CM 1

CRN 7664-93-9
CMF H2 O4 S



CM 2

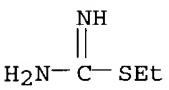
CRN 2986-19-8
CMF C2 H6 N2 S



110 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
111 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 107 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 13882-28-5 REGISTRY
CN Carbamimidothioic acid, ethyl ester, monohydrochloride (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-ethyl-2-thio-, monohydrochloride (8CI)
OTHER NAMES:
CN Carbamimidothioic acid, ethyl ester, hydrochloride
CN Ethiron chloride
CN S-Ethylisothiourea hydrochloride
DR 50292-16-5

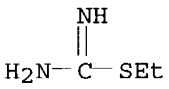
MF C3 H8 N2 S . Cl H
LC STN Files: BEILSTEIN*, CA, CAPLUS, CHEMCATS, IFICDB, IFIPAT, IFIUDB, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
CRN (2986-20-1)



● HCl

17 REFERENCES IN FILE CA (1907 TO DATE)
17 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 108 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 13882-27-4 REGISTRY
CN Carbamimidothioic acid, ethyl ester, monohydriodide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-ethyl-2-thio-, monohydriodide (8CI)
OTHER NAMES:
CN S-Ethylisothiouronium iodide
CN S-Ethylisothiuronium iodide
MF C3 H8 N2 S . H I
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS, IFICDB, IFIPAT, IFIUDB, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
CRN (2986-20-1)



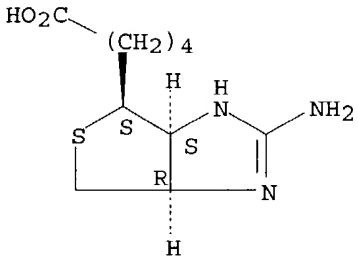
● HI

19 REFERENCES IN FILE CA (1907 TO DATE)
19 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 109 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **13395-35-2** REGISTRY
CN 1H-Thieno[3,4-d]imidazole-4-pentanoic acid, 2-amino-3a,4,6,6a-tetrahydro-, (3aS,4S,6aR)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 1H-Thieno[3,4-d]imidazole-4-pentanoic acid, 2-amino-3a,4,6,6a-tetrahydro-, [3aS-(3aα,4β,6aα)]-
CN Thieno[3,4-d]imidazoline-4-valeric acid, tetrahydro-2-imino- (8CI)
OTHER NAMES:
CN 2-Iminobiotin
CN Iminobiotin
FS STEREOSEARCH
MF C10 H17 N3 O2 S
CI COM

LC STN Files: AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CHEMCATS, CSCHEM, EMBASE, MEDLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

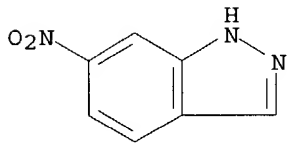
Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

94 REFERENCES IN FILE CA (1907 TO DATE)
19 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
95 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 110 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **7597-18-4** REGISTRY
CN 1H-Indazole, 6-nitro- (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 6-Nitro-1H-indazole
CN 6-Nitroindazole
CN 6-Nitroisoindazole
CN NSC 35066
CN NSC 56816
FS 3D CONCORD
MF C7 H5 N3 O2
CI COM
LC STN Files: BEILSTEIN*, BIOBUSINESS, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DDFU, DRUGU, HODOC*, IFICDB, IFIPAT, IFIUDB, MEDLINE, PIRA, RTECS*, SPECINFO, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

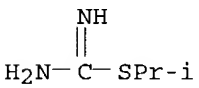


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

170 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
170 REFERENCES IN FILE CAPLUS (1907 TO DATE)

9 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 111 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **6913-17-3** REGISTRY
CN Carbamimidothioic acid, 1-methylethyl ester (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-isopropyl-2-thio- (7CI, 8CI)
OTHER NAMES:
CN 2-Isopropyl-2-thiopseudourea
FS 3D CONCORD
MF C4 H10 N2 S
CI COM
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)



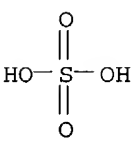
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

19 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
19 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 112 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 6333-32-0 REGISTRY
CN Carbamimidothioic acid, ethyl ester, sulfate (2:1) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-ethyl-2-thio-, sulfate (2:1) (8CI)
OTHER NAMES:
CN Ethylthiouronium sulfate
MF C3 H8 N2 S . 1/2 H2 O4 S
LC STN Files: BEILSTEIN*, CA, CAPLUS
(*File contains numerically searchable property data)

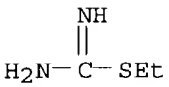
CM 1

CRN 7664-93-9
CMF H2 O4 S



CM 2

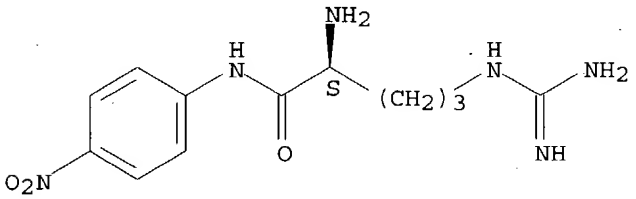
CRN 2986-20-1
CMF C3 H8 N2 S



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 113 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **6154-84-3** REGISTRY
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, (2S)-(9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pentanamide, 2-amino-5-[(aminoiminomethyl)amino]-N-(4-nitrophenyl)-, (S)-
CN Valeranilide, 2-amino-5-guanidino-4'-nitro-, L- (8CI)
OTHER NAMES:
CN Arginine 4-nitroanilide
CN Arginine p-nitroanilide
CN L-Arginine p-nitroanilide
FS STEREOSEARCH
DR 105115-69-3
MF C12 H18 N6 O3
CI COM
LC STN Files: AGRICOLA, BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CSCHEM, EMBASE, PROMT, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.

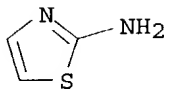


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

61 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
61 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 114 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 6142-05-8 REGISTRY
CN 2-Thiazolamine, monohydrochloride (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Thiazole, 2-amino-, hydrochloride (8CI)
OTHER NAMES:
CN 2-Aminothiazole hydrochloride
CN 2-Thiazolamine hydrochloride
DR 41783-91-9
MF C3 H4 N2 S . Cl H
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CHEMCATS, CHEMLIST, IFICDB, IFIPAT, IFIUDB, TOXCENTER
(*File contains numerically searchable property data)
Other Sources: EINECS**

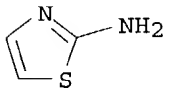
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (96-50-4)



● HCl

36 REFERENCES IN FILE CA (1907 TO DATE)
36 REFERENCES IN FILE CAPLUS (1907 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 115 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 6013-67-8 REGISTRY
CN 2-Thiazolamine, monohydrochloride, monohydrate (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Thiazole, 2-amino-, monohydrochloride, monohydrate (8CI)
OTHER NAMES:
CN 2-Aminothiazole hydrochloride monohydrate
MF C3 H4 N2 S . Cl H . H2 O
CRN (96-50-4)

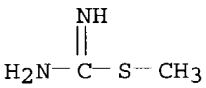


● HCl

● H_2O

L160 ANSWER 116 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 4338-95-8 REGISTRY
CN Carbamimidothioic acid, methyl ester, monohydriodide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-methyl-2-thio-, hydriodide (6CI)
CN Pseudourea, 2-methyl-2-thio-, monohydriodide (8CI)
OTHER NAMES:
CN 2-Methyl-2-isothiourea hydriodide
CN 2-Methyl-2-thiopseudourea hydriodide
CN 2-Methylisothiouronium iodide
CN S-Methylisothiourea hydriodide
CN S-Methylisothiouronium iodide
CN S-Methylisothiuronium iodide
CN S-Methylthiouronium iodide

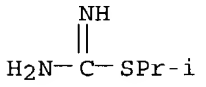
CN S-Methylthiuronium iodide
DR 83893-18-9, 38744-88-6
MF C2 H6 N2 S . H I
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DETHERM*, IFICDB, IFIPAT, IFIUDB, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (2986-19-8)



● HI

108 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
108 REFERENCES IN FILE CAPLUS (1907 TO DATE)
3 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 117 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 4269-97-0 REGISTRY
CN Carbamimidothioic acid, 1-methylethyl ester, monohydrobromide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-isopropyl-2-thio-, hydrobromide (7CI, 8CI)
MF C4 H10 N2 S . Br H
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CSCHEM, TOXCENTER
(*File contains numerically searchable property data)
CRN (6913-17-3)

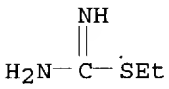


● HBr

10 REFERENCES IN FILE CA (1907 TO DATE)
10 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 118 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **2986-20-1** REGISTRY
CN Carbamimidothioic acid, ethyl ester (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-ethyl-2-thio- (6CI, 7CI, 8CI)
OTHER NAMES:

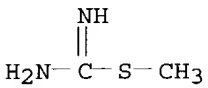
CN 2-Ethyl-2-thiopseudourea
CN S-EIT
CN S-Ethylisothiourea
FS 3D CONCORD
DR 161722-89-0
MF C3 H8 N2 S
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMINFORMRX, CHEMLIST, DDFU, DRUGU, IFICDB, IFIPAT, IFIUDB, MEDLINE, NIOSHTIC, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

96 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
96 REFERENCES IN FILE CAPLUS (1907 TO DATE)
10 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

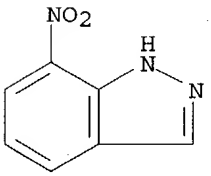
L160 ANSWER 119 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **2986-19-8** REGISTRY
CN Carbamimidothioic acid, methyl ester (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-methyl-2-thio- (6CI, 8CI)
OTHER NAMES:
CN 2-Methyl-2-pseudothiourea
CN 2-Methyl-2-thiopseudourea
CN 2-Methylisothiourea
CN 2-Methylpseudothiourea
CN 2-Methylthiourea
CN Methylisothiourea
CN Methylisothiuronium
CN Methylthiopseudourea
CN S-Methylisothiourea
CN S-Methylpseudothiourea
CN S-Methylthiopseudourea
CN S-Methylthiourea
FS 3D CONCORD
DR 53212-69-4, 72046-62-9
MF C2 H6 N2 S
CI COM
LC STN Files: AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DDFU, DRUGU, HODOC*, IFICDB, IFIPAT, IFIUDB, MEDLINE, NIOSHTIC, RTECS*, SYNTHLINE, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

373 REFERENCES IN FILE CA (1907 TO DATE)
11 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
374 REFERENCES IN FILE CAPLUS (1907 TO DATE)
14 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 120 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **2942-42-9** REGISTRY
CN 1H-Indazole, 7-nitro- (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 7-Nitro-1H-indazole
CN 7-Nitroindazole
CN NSC 72843
FS 3D CONCORD
MF C7 H5 N3 O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, HODOC*, IFICDB, IFIPAT, IFIUDB, MEDLINE, RTECS*, SPECINFO, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)



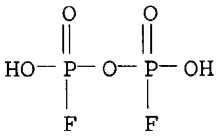
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

338 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
338 REFERENCES IN FILE CAPLUS (1907 TO DATE)
5 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 121 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 2263-91-4 REGISTRY
CN Pseudourea, 2-methyl-2-thio-, P,P'-difluoropyrophosphate (salt) (2:1) (8CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-methyl-2-thio-, P,P'-difluoropyrophosphate (7CI)
MF C2 H6 N2 S . 1/2 F2 H2 O5 P2
LC STN Files: BEILSTEIN*, CAOLD
(*File contains numerically searchable property data)

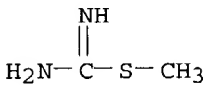
CM 1

CRN 44801-72-1
CMF F2 H2 O5 P2



CM 2

CRN 2986-19-8
CMF C2 H6 N2 S

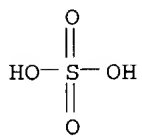


1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 122 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 2260-00-6 REGISTRY
CN Carbamimidothioic acid, methyl ester, sulfate (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-methyl-2-thio-, sulfate (8CI)
OTHER NAMES:
CN S-Methylisothiuronium sulfate
CN S-Methylthiopseudourea sulfate
CN S-Methylthiouronium sulfate
DR 147895-43-0
MF C2 H6 N2 S . x H2 O4 S
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CASREACT, CHEMINFORMRX, CHEMLIST, IFICDB, IFIPAT, IFIUDB, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

CM 1

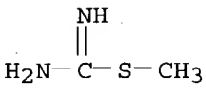
CRN 7664-93-9
CMF H2 O4 S



CM 2

CRN 2986-19-8

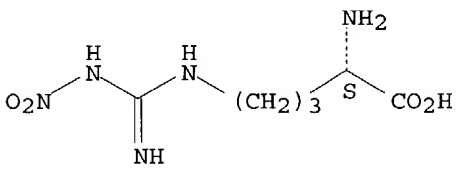
CMF C2 H6 N2 S



204 REFERENCES IN FILE CA (1907 TO DATE)
204 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 123 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **2149-70-4** REGISTRY
CN L-Ornithine, N5-[imino(nitroamino)methyl]- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ornithine, N5-(nitroamidino)-, L- (8CI)
OTHER NAMES:
CN (+)-NG-Nitroarginine
CN ω-Nitro-L-arginine
CN ω-Nitroarginine
CN L-Arginine, ω-nitro-
CN L-Arginine, NG-nitro-
CN L-NG-Nitroarginine
CN L-NNA
CN Nω-Nitro-L-arginine
CN Nω-Nitro-L-arginine
CN NG-Nitro-L-arginine
CN NG-Nitroarginine
CN Nitro-L-arginine
CN Nitroarginine
CN NOLA
CN NSC 53662
FS STEREOSEARCH
DR 13855-78-2, 126265-23-4, 38733-00-5
MF C6 H13 N5 O4
CI COM
LC STN Files: ADISNEWS, AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, PROMT, RTECS*, SPECINFO, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.

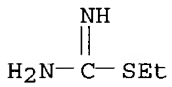


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

946 REFERENCES IN FILE CA (1907 TO DATE)
21 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

946 REFERENCES IN FILE CAPLUS (1907 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 124 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 1071-37-0 REGISTRY
CN Carbamimidothioic acid, ethyl ester, monohydrobromide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-ethyl-2-thio-, hydrobromide (6CI, 7CI)
CN Pseudourea, 2-ethyl-2-thio-, monohydrobromide (8CI)
OTHER NAMES:
CN 2-Ethyl-2-thiopseudourea hydrobromide
CN 2-Ethylisothiourea hydrobromide
CN Ethiron
CN Ethiron bromide
CN Ethyl imidothiocarbamate hydrobromide
CN Etiron
CN Isothuron hydrobromide
CN Isothurone hydrobromide
CN S-Ethylisothiourea hydrobromide
CN S-Ethylisothiouronium hydrobromide
CN S-Ethylisothiuronium bromide
CN S-Ethylthiuronium bromide
CN WR 539
DR 133514-72-4
MF C3 H8 N2 S . Br H
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, IFICDB, IFIPAT, IFIUDB, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
CRN (2986-20-1)

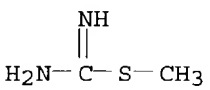


● HBr

177 REFERENCES IN FILE CA (1907 TO DATE)
177 REFERENCES IN FILE CAPLUS (1907 TO DATE)
8 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 125 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 1068-58-2 REGISTRY
CN Carbamimidothioic acid, methyl ester, monohydrobromide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-methyl-2-thio-, monohydrobromide (8CI)
OTHER NAMES:
CN Methiron
CN Methylisothiuronium bromide
CN S-Methylisothiourea hydrobromide
CN S-Methylisothiuronium bromide
CN S-Methylthiouronium bromide

DR 38750-50-4
MF C2 H6 N2 S . Br H
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CASREACT, CHEMLIST, IFICDB, IFIPAT, IFIUDB, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
CRN (2986-19-8)



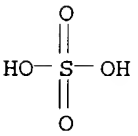
● HBr

16 REFERENCES IN FILE CA (1907 TO DATE)
16 REFERENCES IN FILE CAPLUS (1907 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 126 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 867-44-7 REGISTRY
CN Carbamimidothioic acid, methyl ester, sulfate (2:1) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-methyl-2-thio-, sulfate (2:1) (8CI)
OTHER NAMES:
CN 2-Methyl-2-thiopseudourea sulfate (2:1)
CN Bis(S-methylisothiouronium) sulfate
CN Methylisothiourea sulfate (2:1)
CN Methylthioformamidine sulfate (2:1)
CN S-Methylisothiourea hemisulfate
CN S-Methylisothiourea hemisulfuric acid salt
CN S-Methylisothiourea semisulfate
CN S-Methylisothiourea sulfate (2:1)
CN S-Methylisothiouronium hemisulfate
CN S-Methylthiopseudourea sulfate
CN S-Methylthiouronium sulfate (2:1)
DR 72046-63-0
MF C2 H6 N2 S . 1/2 H2 O4 S
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, IFICDB, IFIPAT, IFIUDB, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

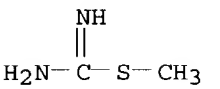
CM 1

CRN 7664-93-9
CMF H2 O4 S



CM 2

CRN 2986-19-8
CMF C2 H6 N2 S

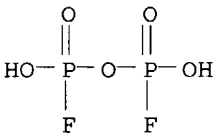


400 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
400 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L160 ANSWER 127 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN 819-15-8 REGISTRY
CN Pseudourea, 2-ethyl-2-thio-, P,P'-difluoropyrophosphate (2:1) (8CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Pseudourea, 2-ethyl-2-thio-, fluopyrophosphate (6CI)
CN Pseudourea, 2-ethyl-2-thio-, P,P'-difluoropyrophosphate (7CI)
MF C3 H8 N2 S . 1/2 F2 H2 O5 P2
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS
(*File contains numerically searchable property data)

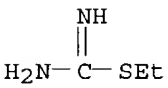
CM 1

CRN 44801-72-1
CMF F2 H2 O5 P2



CM 2

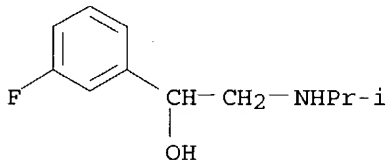
CRN 2986-20-1
CMF C3 H8 N2 S



1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 128 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **451-04-7** REGISTRY
CN Benzenemethanol, 3-fluoro-α-[[(1-methylethyl)amino]methyl]- (9CI) (CA INDEX NAME)

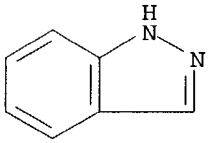
OTHER CA INDEX NAMES:
CN Benzyl alcohol, m-fluoro-α-[(isopropylamino)methyl]- (6CI, 8CI)
FS 3D CONCORD
MF C11 H16 F N O
CI COM
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, TOXCENTER
(*File contains numerically searchable property data)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

5 REFERENCES IN FILE CA (1907 TO DATE)
5 REFERENCES IN FILE CAPLUS (1907 TO DATE)
4 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

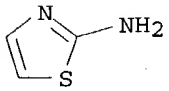
L160 ANSWER 129 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **271-44-3** REGISTRY
CN 1H-Indazole (7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Indazole (6CI)
OTHER NAMES:
CN 1,2-Diazaindene
CN 1H-Benzopyrazole
CN 2-Azaindole
CN Isoindazole
CN NSC 26336
CN NSC 90357
FS 3D CONCORD
DR 116421-37-5
MF C7 H6 N2
CI COM, RPS
LC STN Files: AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DETHERM*, EMBASE, GMELIN*, HODOC*, IFICDB, IFIPAT, IFIUDB, MRCK*, NIOSHTIC, PIRA, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

791 REFERENCES IN FILE CA (1907 TO DATE)
57 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
792 REFERENCES IN FILE CAPLUS (1907 TO DATE)
33 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L160 ANSWER 130 OF 130 REGISTRY COPYRIGHT 2004 ACS on STN
RN **96-50-4** REGISTRY
CN 2-Thiazolamine (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Thiazole, 2-amino- (8CI)
OTHER NAMES:
CN 2-Amino-1,3-thiazole
CN 2-Aminothiazole
CN 2-Thiazolylamine
CN Abadol
CN Abadole
CN Aminothiazole
CN Basedol
CN NSC 1900
FS 3D CONCORD
DR 58473-79-3, 5654-01-3
MF C3 H4 N2 S
CI COM
LC STN Files: ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DETHERM*, DRUGU, EMBASE, GMELIN*, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1909 REFERENCES IN FILE CA (1907 TO DATE)
65 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1915 REFERENCES IN FILE CAPLUS (1907 TO DATE)
15 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> d his

(FILE 'HOME' ENTERED AT 07:16:13 ON 03 MAR 2004)

FILE 'REGISTRY' ENTERED AT 07:16:54 ON 03 MAR 2004

L1 (12)SEA FILE=REGISTRY ABB=ON PLU=ON 180001-34-7 OR 168895-09-8 OR
L2 (11)SEA FILE=REGISTRY ABB=ON PLU=ON 36889-13-1 OR 30315-93-6 OR 2
L3 (2)SEA FILE=REGISTRY ABB=ON PLU=ON 2986-20-1 OR 2986-19-8
L4 25 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3)
L5 (18)SEA FILE=REGISTRY ABB=ON PLU=ON (139299-32-4/CRN OR 139461-37
L6 (153)SEA FILE=REGISTRY ABB=ON PLU=ON (13395-35-2/CRN OR 17035-90-4
L7 (200)SEA FILE=REGISTRY ABB=ON PLU=ON (2986-19-8/CRN OR 2986-20-1/C

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L8          371 SEA FILE=REGISTRY ABB=ON  PLU=ON  (L5 OR L6 OR L7)
L9            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  10102-43-9/RN
L10           4 S 40127-14-8 OR 6154-84-3 OR 140845-10-9 OR 40127-15-9
L11           7 S (40127-14-8 OR 6154-84-3 OR 140845-10-9 OR 40127-15-9)/CRN
L12          29 S L4 OR L10
L13         378 L8 OR L11
L14         101 S L13 NOT ((PMS OR IDS)/CI OR UNS OR COMPD)

     FILE 'HCAPLUS' ENTERED AT 07:27:06 ON 03 MAR 2004
L15        6680 S L12 OR L14
                E PRURITIS/CT
                E E4+ALL
L16        1454 S PRURITUS + OLD/CT
L17       13795 SKIN, DISEASE/CT
L18           5 L17 (L) PRURIGO NODULARIS
L19           2 L17 (L) PRURIGO PIGMENTOSA
L20          37 S L17 (L) (ITCH? OR SCRATCH? OR RUBBING?)
L21         336 (PRURIT? OR ITCH? OR SCRATCH?) (L) (DISEASE? OR DISORDER?)
L22        1540 PRURIT?
L23        9022 ITCH? OR SCRATCH? OR RUBBING?
L24           5 S L15 AND (L16 OR L18-23)
L25           4 S L24 NOT SILVER HALIDE/TI
L26      140433 (OXOAMIDOGEN OR OXO AMIDOGEN OR INOMAX OR (NITRIC OR NITROGEN?)
L27          35 1400W OR 1400 W OR W1400 OR W 1400
L28           4 ARR17477 OR ARR17 477 OR ARR 17477 OR ARR 17 477 OR AR R 17477
L29           6 S ETHYLCITRULLINE OR S ETHYL L THIOCITRULLINE OR S ETHYL THIOCI
L30           2 1 3 PBIT
L31           2 S METHYL L CITRULLINE OR S METHYLCITRULLINE OR S MTC
L32           0 L HOMOTIOCITRULLINE
L33           4 (N OMEGA OR NW) () ALLYL L ARGININE
L34           0 (N OMEGA OR NW) () CYCLOPROPYL L ARGININE
L35           2 (N OMEGA OR NW) () AMINO L ARGININE
L36         113 2 AMINOTHIAZOLINE
L37        4382 L NAME OR (N OR N OMEGA OR NG) () (ME OR METHYL) () ESTER OR NA
L38           2 L NIO OR N DELTA IMINOETHYL L ORTHININE OR N5 () (1 IMINOETHYL)
L39         229 ADMA OR (ASYMMETRIC OR L NG NG OR NG NG) () DIMETHYLARGININE OR
L40          10 (2 TRIFLUOROMETHYL) () IMIDAZOLE
L41         662 (OMEGA N OR NG)()(METHLARGININE OR MEONOMETHYLARGININE) OR L()(
L42          88 2 IMINIBIOTIN OR IMINOBIOTIN
L43          49 L () (TC OR THIOCITRULLINE)
L44           8 L () (NIL OR EPSILON ACETIMIDYLLSINE)
L45           9 6 NITRO!!!INDAZOLE OR 6 NITRO 1H INDAZOLE OR NSC35066 OR NSC568
L46           1 2 ISOPROPYL 2 THIOPSEUDOUREA
L47          61 2 ETHYL 2 THIOPSEUDOUREA OR S ()(ETHYLISOTHIOUREA OR EIT)
L48         752 (2 OR S) () (METHYL () (PSEUDOTHIOUREA OR THIOPSEUDOUREA) OR ME
L49         284 7 NITROINDAZOLE OR 7 NITRO 1H INDAZOLE OR NSC72843 OR NSC 72843
L50        1406 NSC53662 OR NSC 53662 OR NSC 52 662 OR (OMEGA OR N OMEGA OR NG)
L51        2721 INDA!OLE OR ISOINDA!OLE OR 2 AZAINDOLE OR 1 2 DIAZAINDENE OR 1H
L52        2450 2 () (AMINO 1 3 THIAZOLE OR AMINOTHIAZOLE OR THIAZOYLAMINE) OR
L53           1 ARGININE () (P NITROANILIDE) () DIHYDROFLUORIDE
L54           0 N OMEGA D ARGININE P NITROANILIDE HYDROBROMIDE
L55           0 N OMEGA NITRO DL ARGININE P NITROANILIDE HYDROBROMIDE
L56         127 ARGININE () ((4 OR P) () NITROANILIDE) OR L ARGININE P NITROANI
L57        2660 S L26 AND L27-56
L58           3 S L57 AND (L16 OR L18-23)

     FILE 'REGISTRY' ENTERED AT 08:42:07 ON 03 MAR 2004
L59           1 451-04-7

     FILE 'HCAPLUS' ENTERED AT 08:42:09 ON 03 MAR 2004
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L60          20 NITRIC OXIDE MODULATOR
L61           5 S L59
L62           0 S L60-61 AND (L16 OR L18-23)

     FILE 'MEDLINE' ENTERED AT 08:48:01 ON 03 MAR 2004
L63        7140 S L12
L64           1 S L14
L65       61399 (OXOAMIDOGEN OR OXO AMIDOGEN OR INOMAX OR (NITRIC OR NITROGEN?)
L66         147 1400W OR 1400 W OR W1400 OR W 1400
L67           7 ARR17477 OR ARR17 477 OR ARR 17477 OR ARR 17 477 OR AR R 17477
L68           1 S ETHYLCITRULLINE OR S ETHYL L THIOCITRULLINE OR S ETHYL THIOCI
L69           0 1 3 PBIT
L70           3 S METHYL L CITRULLINE OR S METHYLCITRULLINE OR S MTC
L71           0 L HOMOTIOCITRULLINE
L72          92 L () (TC OR THIOCITRULLINE)
L73           2 (N OMEGA OR NW) () ALLYL L ARGININE
L74           0 (N OMEGA OR NW) () CYCLOPROPYL L ARGININE
L75           5 (N OMEGA OR NW) () AMINO L ARGININE
L76         146 L () (NIL OR EPSILON ACETIMIDYLLSINE)
L77          28 2 AMINOTHIAZOLINE
L78       23767 L NAME OR (N OR N OMEGA OR NG) () (ME OR METHYL) () ESTER OR NA
L79         100 L NIO OR N DELTA IMINOETHYL L ORTHININE OR N5 () (1 IMINOETHYL)
L80         336 ADMA OR (ASYMMETRIC OR L NG NG OR NG NG) () DIMETHYLARGININE OR
L81           2 (2 TRIFLUOROMETHYL) () IMIDAZOLE
L82        4701 (OMEGA N OR NG)()(METHLARGININE OR MEONOMETHYLARGININE) OR L()(
L83          51 2 IMINIBIOTIN OR IMINOBIOTIN
L84           0 6 NITRO!!!INDAZOLE OR 6 NITRO 1H INDAZOLE OR NSC35066 OR NSC568
L85           0 2 ISOPROPYL 2 THIOPSEUDOUREA
L86          34 2 ETHYL 2 THIOPSEUDOUREA OR S ()(ETHYLISOTHIOUREA OR EIT)
L87         165 (2 OR S) () (METHYL () (PSEUDOTHIOUREA OR THIOPSEUDOUREA) OR ME
L88         664 7 NITROINDAZOLE OR 7 NITRO 1H INDAZOLE OR NSC72843 OR NSC 72843
L89       13082 NSC53662 OR NSC 53662 OR NSC 52 662 OR (OMEGA OR N OMEGA OR NG)
L90         373 INDA!OLE OR ISOINDA!OLE OR 2 AZAINDOLE OR 1 2 DIAZAINDENE OR 1H
L91         250 2 () (AMINO 1 3 THIAZOLE OR AMINOTHIAZOLE OR THIAZOYLAMINE) OR
L92           0 ARGININE () (P NITROANILIDE) () DIHYDROFLUORIDE
L93           0 N OMEGA D ARGININE P NITROANILIDE HYDROBROMIDE
L94           0 N OMEGA NITRO DL ARGININE P NITROANILIDE HYDROBROMIDE
L95         416 ARGININE () ((4 OR P) () NITROANILIDE) OR L ARGININE P NITROANI
L96           0 PRURITIS+NT/CT
L97       16840 ANTIPRURITICS+NT/CT
L98       17228 S L21-23
L99       35140 S L9
L100       5811 S PRURITUS+NT/CT
L101          0 S L59
L102          9 S L60
L103      16974 S (L99 OR L65) AND (L63-64 OR L66-95 OR L101-102)
L104         65 S L103 AND (L100 OR L97-98)
L105       8511 S L97/MAJ
L106       2966 S L100/MAJ
L107         47 S L103 AND (L98 OR L105-106)

     FILE 'HCAPLUS' ENTERED AT 09:04:49 ON 03 MAR 2004
L108         19 S L57 AND L17

     FILE 'EMBASE' ENTERED AT 09:11:55 ON 03 MAR 2004
L109      14923 S E3-E25
L110      16541 S E4-16
L111      24381 S L21-23 OR ANTIPRURIT? OR ANTI PRURIT?
L112      42101 S L9
L113       8400 S L12 OR L14 OR L59
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L114       65223 (OXOAMIDOGEN OR OXO AMIDOGEN OR INOMAX OR (NITRIC OR NITROGEN?)
L115         155 1400W OR 1400 W OR W1400 OR W 1400
L116           8 ARR17477 OR ARR17 477 OR ARR 17477 OR ARR 17 477 OR AR R 17477
L117           1 S ETHYLCITRULLINE OR S ETHYL L THIOCITRULLINE OR S ETHYL THIOCI
L118           0 1 3 PBIT
L119           3 S METHYL L CITRULLINE OR S METHYLCITRULLINE OR S MTC
L120           0 L HOMOTIOCITRULLINE
L121          87 L () (TC OR THIOCITRULLINE)
L122           3 (N OMEGA OR NW) () ALLYL L ARGININE
L123           0 (N OMEGA OR NW) () CYCLOPROPYL L ARGININE
L124           5 (N OMEGA OR NW) () AMINO L ARGININE
L125         129 L () (NIL OR EPSILON ACETIMIDYLLSINE)
L126          12 2 AMINOTHIAZOLINE
L127       19856 L NAME OR (N OR N OMEGA OR NG) () (ME OR METHYL) () ESTER OR NA
L128          97 L NIO OR N DELTA IMINOETHYL L ORTHININE OR N5 () (1 IMINOETHYL)
L129         267 ADMA OR (ASYMMETRIC OR L NG NG OR NG NG) () DIMETHYLARGININE OR
L130           2 (2 TRIFLUOROMETHYL) () IMIDAZOLE
L131        4891 (OMEGA N OR NG)()(METHLARGININE OR MEONOMETHYLARGININE) OR L()(
L132          36 2 IMINIBIOTIN OR IMINOBIOTIN
L133           1 6 NITRO!!!INDAZOLE OR 6 NITRO 1H INDAZOLE OR NSC35066 OR NSC568
L134           0 2 ISOPROPYL 2 THIOPSEUDOUREA
L135          35 2 ETHYL 2 THIOPSEUDOUREA OR S ()(ETHYLISOTHIOUREA OR EIT)
L136         204 (2 OR S) () (METHYL () (PSEUDOTHIOUREA OR THIOPSEUDOUREA) OR ME
L137         740 7 NITROINDAZOLE OR 7 NITRO 1H INDAZOLE OR NSC72843 OR NSC 72843
L138       13380 NSC53662 OR NSC 53662 OR NSC 52 662 OR (OMEGA OR N OMEGA OR NG)
L139         941 INDA!OLE OR ISOINDA!OLE OR 2 AZAINDOLE OR 1 2 DIAZAINDENE OR 1H
L140         389 2 () (AMINO 1 3 THIAZOLE OR AMINOTHIAZOLE OR THIAZOYLAMINE) OR
L141           0 ARGININE () (P NITROANILIDE) () DIHYDROFLUORIDE
L142           0 N OMEGA D ARGININE P NITROANILIDE HYDROBROMIDE
L143           0 N OMEGA NITRO DL ARGININE P NITROANILIDE HYDROBROMIDE
L144         255 ARGININE () ((4 OR P) () NITROANILIDE) OR L ARGININE P NITROANI
L145           6 NITRIC OXIDE MODULATOR
L146       16594 S (L112 OR L114) AND (L113 OR L115-L145)
L147          52 S L146 AND L109-111
L148          44 S L147 AND PD<20011017

     FILE 'WPIX' ENTERED AT 09:37:31 ON 03 MAR 2004
                E B14-N17/MC
L149       16986 S E3
                E B12-A07/MC
L150       13388 S E3
L151        2133 S C12-A07/MC
L152        1178 S C14-N17/MC
                E A61P017-04/IC
L153         512 S E3

     FILE 'WPIX' ENTERED AT 09:41:56 ON 03 MAR 2004
L154          19 S A61P017-04/ICA,ICI
L155        1587 S B05-C03/MC
L156        1259 S C05-C03/MC
L157        5150 S (E31H OR E31-H02)/MC
L158        2895 S E31-H/MC

     FILE 'REGISTRY' ENTERED AT 09:51:29 ON 03 MAR 2004
L159         129 S L12 OR L14
L160         130 S L12 OR L14 OR L59

     FILE 'HCAPLUS' ENTERED AT 09:53:36 ON 03 MAR 2004
L161          23 S L25 OR L58 OR L108
                 SAVE TEMP MOH380HCAP/A L161
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     FILE 'MEDLINE' ENTERED AT 09:54:26 ON 03 MAR 2004
                SAVE TEMP L107 MOH380MED/A

     FILE 'EMBASE' ENTERED AT 09:55:16 ON 03 MAR 2004
                SAVE TEMP L148 MOH380EMB/A

     FILE 'WPIX' ENTERED AT 10:06:10 ON 03 MAR 2004
L162        48109 S C107/M0,M1,M2,M3,M4,M5,M6
L163       282662 S C108/M0,M1,M2,M3,M4,M5,M6
L164        27614 S L162 (S) L163
                  E NO/CN
                  E E3+ALL
L165            1 S E3
L166        20679 ((OXOAMIDOGEN OR OXO AMIDOGEN OR INOMAX OR (NITRIC OR NITROGEN?
L167           20 (1400W OR 1400 W OR W1400 OR W 1400)/BIX
L168            0 (ARR17477 OR ARR17 477 OR ARR 17477 OR ARR 17 477 OR AR R 17477
L169            2 (S ETHYLCITRULLINE OR S ETHYL L THIOCITRULLINE OR S ETHYL THIOC
L170            5 (1 3 PBIT)/BIX
L171            1 (S METHYL L CITRULLINE OR S METHYLCITRULLINE OR S MTC)/BIX
L172            0 (L HOMOTIOCITRULLINE)/BIX
L173           19 (L () (TC OR THIOCITRULLINE))/BIX
L174            1 ((N OMEGA OR NW) () ALLYL L ARGININE)/BIX
L175            1 ((N OMEGA OR NW) () CYCLOPROPYL L ARGININE)/BIX
L176            1 ((N OMEGA OR NW) () AMINO L ARGININE)/BIX
L177            8 (L () (NIL OR EPSILON ACETIMIDYLLSINE))/BIX
L178           34 (2 AMINOTHIAZOLINE)/BIX
L179        27824 (L NAME OR (N OR N OMEGA OR NG) () (ME OR METHYL) () ESTER OR N
L180            6 (L NIO OR N DELTA IMINOETHYL L ORTHININE OR N5 () (1 IMINOETHYL
L181           28 (ADMA OR (ASYMMETRIC OR L NG NG OR NG NG) () DIMETHYLARGININE O
L182            2 ((2 TRIFLUOROMETHYL) () IMIDAZOLE)/BIX
L183           71 ((OMEGA N OR NG)()(METHLARGININE OR MEONOMETHYLARGININE) OR L()
L184           45 (2 IMINIBIOTIN OR IMINOBIOTIN)/BIX
L185            0 (6 NITRO!!!INDAZOLE OR 6 NITRO 1H INDAZOLE OR NSC35066 OR NSC56
L186            0 (2 ISOPROPYL 2 THIOPSEUDOUREA)/BIX
L187          132 ((2 OR S) () (METHYL () (PSEUDOTHIOUREA OR THIOPSEUDOUREA) OR M
L188           22 (7 NITROINDAZOLE OR 7 NITRO 1H INDAZOLE OR NSC72843 OR NSC 7284
L189          127 (NSC53662 OR NSC 53662 OR NSC 52 662 OR (OMEGA OR N OMEGA OR NG
L190          916 (INDA!OLE OR ISOINDA!OLE OR 2 AZAINDOLE OR 1 2 DIAZAINDENE OR 1
L191          841 (2 () (AMINO 1 3 THIAZOLE OR AMINOTHIAZOLE OR THIAZOYLAMINE) OR
L192            0 (ARGININE () (P NITROANILIDE) () DIHYDROFLUORIDE)/BIX
L193            0 (N OMEGA D ARGININE P NITROANILIDE HYDROBROMIDE)/BIX
L194            0 (N OMEGA NITRO DL ARGININE P NITROANILIDE HYDROBROMIDE)/BIX
L195           26 (ARGININE () ((4 OR P) () NITROANILIDE) OR L ARGININE P NITROAN
L196            3 (NITRIC OXIDE MODULATOR)/BIX
L197           14 (2 ETHYL 2 THIOPSEUDOUREA OR S ()(ETHYLISOTHIOUREA OR EIT))/BIX
L198        35352 ((PRURIT? OR ITCH? OR SCRATCH?) (L) (DISEASE? OR DISORDER?) OR
L199          159 S (L155-158 OR L164-166) AND L167-196
L200           14 S L199 AND (L198 OR L149-154)

=> b hcap

FILE 'HCAPLUS' ENTERED AT 10:45:30 ON 03 MAR 2004
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FILE COVERS 1907 - 3 Mar 2004 VOL 140 ISS 10
FILE LAST UPDATED: 2 Mar 2004 (20040302/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

'OBI' IS DEFAULT SEARCH FIELD FOR 'HCAPLUS' FILE

=> d all l161 tot

L161 ANSWER 1 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:166719 HCAPLUS
DN 139:111508
ED Entered STN: 05 Mar 2003
TI Systemic agmatine attenuates tactile allodynia in two experimental neuropathic pain models in rats
AU Karadag, Hakan C.; Ulugol, Ahmet; Tamer, Melek; Ipci, Yesim; Dokmeci, Ismet
CS Faculty of Medicine, Department of Pharmacology, Trakya University, Edirne, 22030, Turk.
SO Neuroscience Letters (2003), 339(1), 88-90
CODEN: NELED5; ISSN: 0304-3940
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
CC 1-11 (Pharmacology)
AB This study evaluated the effect of agmatine on allodynia in two exptl. neuropathic pain models: the spinal nerve ligation model and streptozotocin-induced diabetic neuropathy in rats; the study also investigated whether N-methyl-D-aspartate (NMDA) receptor antagonists and NO synthase (NOS) inhibitors would influence this effect of agmatine. Nerve injury was produced by tight ligation of the left L5 and L6 spinal nerves, and diabetic neuropathy was induced by the injection of a single dose of streptozotocin; these procedures resulted in tactile allodynia in the hindpaw. Tactile allodynia was detected by application of von Frey filaments to the plantar surface of the foot. Agmatine at the higher doses tested reduced mech. allodynia. Dizocilpine maleate, an NMDA receptor antagonist, and the NOS inhibitors NG-nitro-L-arginine Me ester and 7-nitroindazole did not influence the antiallodynic effect of agmatine. These results suggest that agmatine has an antiallodynic effect in both spinal nerve ligation and diabetic models and may be a promising drug in the treatment of neuropathic pain.
ST agmatine allodynia inhibition neuropathic pain; analgesia agmatine
IT Glutamate antagonists
(NMDA antagonists; agmatine attenuation of tactile allodynia in neuropathic pain response to)
IT Analgesics
(agmatine attenuation of tactile allodynia in two exptl. neuropathic pain models)
IT Pain
Skin, disease

(allodynia; agmatine attenuation of tactile allodynia in two exptl. neuropathic pain models)
IT Nerve, disease
(diabetic neuropathy; agmatine attenuation of tactile allodynia in two exptl. neuropathic pain models)
IT Pain
(neuropathic; agmatine attenuation of tactile allodynia in two exptl. neuropathic pain models)
IT 2942-42-9, **7-Nitroindazole** 50903-99-6, N-**Nitro-L-arginine** methyl ester 77086-22-7, Dizocilpine maleate
RL: PAC (Pharmacological activity); BIOL (Biological study)
(agmatine attenuation of tactile allodynia in neuropathic pain response to)
IT 306-60-5, Agmatine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(agmatine attenuation of tactile allodynia in two exptl. neuropathic pain models)
IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibitors; agmatine attenuation of tactile allodynia in neuropathic pain response to)
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Begon, S; Brain Res 2000, V887, P436 HCAPLUS
(2) Calcutt, N; Br J Pharmacol 1997, V122, P1478 HCAPLUS
(3) Chaplan, S; J Neurosci Methods 1994, V53, P55 MEDLINE
(4) Courteix, C; Pain 1993, V53, P81 MEDLINE
(5) Dixon, W; Ann Rev Pharmacol Toxicol 1980, V20, P441 MEDLINE
(6) Esser, M; Pain 1999, V80, P643 HCAPLUS
(7) Fairbanks, C; PNAS 2000, V97, P10584 HCAPLUS
(8) Galea, E; Biochem J 1996, V316, P247 HCAPLUS
(9) Kim, S; Pain 1992, V50, P355 MEDLINE
(10) Kolesnikov, Y; Eur J Pharmacol 1996, V296, P17 HCAPLUS
(11) Lee, Y; Neurosci Lett 1995, V186, P1
(12) MacFarlane, B; Pharmacol Ther 1997, V75, P1 HCAPLUS
(13) Onal, A; Pharmacol Biochem Behav 2001, V69, P93 HCAPLUS
(14) Piletz, J; J Pharmacol 1995, V272, P581 HCAPLUS
(15) Reis, D; TiPS 2000, V9, P187
(16) Ulugol, A; Neurosci Lett 2002, V328, P129 HCAPLUS
(17) Ulugol, A; Neurosci Res Commun 2002, V30, P143 HCAPLUS
(18) Yaksh, T; J Pharmacol Exp Ther 1995, V272, P207 HCAPLUS
(19) Yang, X; J Pharmacol Exp Ther 1999, V288, P544 HCAPLUS
(20) Yesilyurt, O; Neuropsychopharmacology 2001, V25, P98 HCAPLUS

L161 ANSWER 2 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:41926 HCAPLUS
DN 138:78199
ED Entered STN: 17 Jan 2003
TI Cosmetic and dermatological preparations containing carnitine, acylcarnitines and NO-synthase inhibitors
IN Mummert, Christopher; Mundt, Claudia; Blatt, Thomas; Kolbe, Ludger; Schoenrock, Uwe
PA Beiersdorf A.-G., Germany
SO Ger. Offen., 22 pp.
CODEN: GWXXBX
DT Patent
LA German

IC ICM A61K007-48
CC 62-4 (Essential Oils and Cosmetics)
Section cross-reference(s): 63
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 10129503	A1	20030116	DE 2001-10129503	20010619
PRAI	DE 2001-10129503		20010619		

AB The invention concerns cosmetic and dermatol. prepns. that contain carnitine, acylcarnitine and NO-synthase inhibitors, preferably nitroarginine for the prevention and treatment of dry skin, skin pigmentation, for increasing ceramide biosynthesis, for enhancing skin barrier function and use as sunscreens. Thus an O/W cream contained (weight/weight%): glyceryl stearate 3.00; stearic acid 1.00; cetyl alc. 2.00; dicapryl ether 4.00; caprylic acid/caprylic acid triglyceride 3.00; paraffin oil 2.00; glycerin 3.00; butylene glycol 3.00; carbomer 0.10; carnitine 1.00; NG-nitro-L-arginine 0.10; sodium hydroxide, preservatives, perfume q.s.; water to 100.
ST skin cream carnitine acylcarnitine NO synthase inhibitor **nitroarginine**
IT Sunscreens
(cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
IT Cosmetics
(creams; cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
IT **Skin, disease**
(depigmentation; cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
IT **Skin, disease**
(dry; cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
IT Ceramides
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(increasing biosynthesis of; cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
IT Cosmetics
(lotions; cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
IT Cosmetics
(makeups; cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
IT 541-15-1, Carnitine 541-15-1D, Carnitine, esters 2149-70-4
RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; cosmetic and dermatol. prepns. containing carnitine, acylcarnitine and NO-synthase inhibitors)
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Anon; DE 19806889 A1 HCAPLUS
(2) Anon; DE 19806890 A1 HCAPLUS
(3) Anon; DE 19806946 A1 HCAPLUS
(4) Anon; DE 19806947 A1 HCAPLUS
(5) Anon; DE 19918750 A1 HCAPLUS

L161 ANSWER 3 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN

AN 2002:831736 HCAPLUS
DN 137:329273
ED Entered STN: 01 Nov 2002
TI Cosmetic and dermatological skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides
IN Mundt, Claudia; Kolbe, Ludger; Kroepke, Rainer; Mummert, Christopher; Wolber, Rainer
PA Beiersdorf AG, Germany
SO Ger. Offen., 22 pp.
CODEN: GWXXBX
DT Patent
LA German
IC ICM A61K007-48
CC 62-4 (Essential Oils and Cosmetics)
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 10121089	A1	20021031	DE 2001-10121089	20010426
PRAI DE 2001-10121089		20010426		

AB The invention concerns cosmetic and dermatol. skin formulations that contain substances that inhibit the activity of NO synthase or inhibit the expression of the enzyme; the formulations further contain tetrahydrocurcumin, tetrahydrodemethoxycurcumin, and/or tetrahydrobisdemethoxycurcumin. The compns. are used to prevent skin dryness and pigmentation, to stimulate ceramide synthesis and as sunscreens. Creams, lotions and sprays are formulated. Thus a spray contained (weight/weight%): glycerin monostearate SE 0.50; Ceteareth -30 5.00; cetyl alc. 2.50; dioctyl butamidotriazone 1.00; ethylhexyl triazone 4.00; phenylbenzimidazole sulfonic acid 0.50; titanium dioxide 0.0; butylene glycol dicaprylate/dicaprate 5.00; phenyltrimethicone 2.00; PVP hexadecene copolymer 0.50; glycerin 3.00; Vitamin E acetate 0.50; tetrahydrocurcumin 0.20; nitroguanidine 0.10; guanidino glutaric acid 0.20; α-glucosylrutin 0.10; DMDM hydantoin 0.60; phenoxyethanol 0.50; ethanol 3.00; perfume q.s.; water to 100.

ST NO synthase inhibitor tetrahydrocurcuminoide cosmetics skin

IT Sunscreens
(cosmetic and dermatol. skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides)

IT Ceramides
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cosmetic and dermatol. skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides)

IT Cosmetics
(creams; cosmetic and dermatol. skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides)

IT Cosmetics
(emulsions; cosmetic and dermatol. skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides)

IT **Skin, disease**
(pigmentation; cosmetic and dermatol. skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides)

IT Cosmetics
(sprays; cosmetic and dermatol. skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides)

IT 50-02-2, Dexamethasone 55-21-0, Benzamide 106-51-4, 2,5-Cyclohexadiene-1,4-dione, biological studies 123-78-4D, Sphingosine, acyl derivs. 244-54-2, Dibenziodolium 244-63-3, Norharman 303-98-0, Ubiquinone Q10 372-75-8, Citrulline 504-29-0, 2-Aminopyridine 543-38-4, Canavanin 556-88-7, Nitroguanidine 695-34-1, 2-Amino-4-Methylpyridine 996-19-0 2149-70-4, **Nitroarginine**

2214-67-7D, 5-hetero derivative 2582-07-2, (2-Benzothiazolyl)guanidine
2942-42-9 2986-19-8, **S-Methylisothiourea** 4269-97-0
4364-78-7, 1,3-Diaminoguanidine 4673-26-1 5154-02-9 6960-42-5,
7-Nitroindole 17035-90-4 19045-66-0, Thiocarbamic acid 21463-31-0
22780-54-7 25371-96-4 31137-74-3 36062-04-1, Tetrahydrocurcumin
36889-13-1 39123-82-5 41137-86-4, Hirsutanonol 41443-28-1
51298-62-5 55303-93-0, Oregonin 60940-34-3, Ebselen 65005-57-4
73477-53-9, α-Guanidino glutaric acid 74209-34-0 113482-94-3,
3,5-Heptanedione, 1,7-bis(4-hydroxyphenyl)- 148819-94-7 149579-07-7,
3,5-Heptanedione, 1-(4-hydroxy-3-methoxyphenyl)-7-(4-hydroxyphenyl)-
150403-89-7 156719-39-0 157254-60-9 159190-45-1 167423-51-0
200716-66-1 211183-26-5
RL: COS (Cosmetic use); BIOL (Biological study); USES (Uses)
(cosmetic and dermatol. skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides)

IT 125978-95-2, Synthase, **nitric oxide**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors of; cosmetic and dermatol. skin formulations containing NO synthase inhibitors and tetrahydrocurcuminoides)

RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE
(1) Anon; WO 0013661 A1 HCAPLUS
(2) Anon; WO 0061162 A1 HCAPLUS
(3) Anon; JP 02049747 A HCAPLUS
(4) Anon; JP 02069431 A HCAPLUS
(5) Anon; JP 06128133 A HCAPLUS
(6) Anon; JP 11235192 A HCAPLUS
(7) Anon; US 5266344 A HCAPLUS
(8) Gupta, S; HAPPI 2001, V38(4), PS90
(9) Lin, J; BioFactors 2000, V13, PS153
(10) Pan, M; Biochemical Pharmacology 2000, V60, PS1665

L161 ANSWER 4 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:695732 HCAPLUS
DN 137:221758
ED Entered STN: 13 Sep 2002
TI Use of subtances that prevent the effects of NO synthase in cosmetic preparations for the prophylaxis and treatment of skin pigmentation disorders
IN Mummert, Christopher; Mundt, Claudia; Blatt, Thomas; Kolbe, Ludger; Wolber, Rainer; Schaumann, Ernst
PA Beiersdorf AG, Germany
SO PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DT Patent
LA German
IC ICM A61K007-00
CC 62-3 (Essential Oils and Cosmetics)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002069910	A2	20020912	WO 2002-EP2120	20020228
	WO 2002069910	A3	20021219		
	W: JP, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
	DE 10111050	A1	20020912	DE 2001-10111050	20010306
PRAI	DE 2001-10111050	A	20010306		

AB The invention concerns substances that inhibit NO synthase activity or block the expression of NO synthase in warm blooded organisms and their

application in cosmetic and dermatol. prepns. for the prevention and treatment of undesired skin pigmentation. Thus an O/W cream contained (weight/weight%): glycerylstearate 4.00; PEG-40-stearate 1.00; cetyl alc. 3.00; caprylic acid/caprylic acid triglyceride 5.00; mineral oil 5.00; nitroguanidine 0.10; tocopherol 0.10; trisodium EDTA 0.10; preservatives q.s.; carbomer 3.00; sodium hydroxide (45%) q.s.; glycerin 5.00; perfume q.s.; water to 100.

ST NO synthase inhibition skin cosmetics pigmentation disorder

IT Cosmetics
(creams; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin pigmentation disorders)

IT Cosmetics
(emulsions; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin pigmentation disorders)

IT **Skin, disease**
(pigmentation; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin pigmentation disorders)

IT Ubiquinones
RL: COS (Cosmetic use); BIOL (Biological study); USES (Uses)
(use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin pigmentation disorders)

IT 125978-95-2, Synthase, **nitric oxide**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors of; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin pigmentation disorders)

IT 50-02-2, Dexamethasone 55-21-0, Benzamide 62-56-6, Thiourea, biological studies 106-51-4, 2,5-Cyclohexadiene-1,4-dione, biological studies 123-78-4D, Sphingosine, N-Acyl derivs. 143-37-3D, Acetamidine, fluorinated, amino acid and oleic acid derivs. 244-54-2, Dibenziodolium 303-98-0, Coenzyme Q10 372-75-8, Citrulline 458-37-7 504-29-0, 2-Aminopyridine 543-38-4, Canavanin 695-34-1, 2-Amino-4-Methylpyridine 996-19-0 2149-70-4 2214-67-7D, hetero derivs. 2942-42-9 2986-19-8, **S-Methylisothiourea** 3416-24-8, D-Glucosamine 4269-97-0 4673-26-1 5154-02-9 17035-90-4 21463-31-0 22780-54-7, 2-Iminopiperidine 25371-96-4 31137-74-3 36889-13-1 41443-28-1 51298-62-5 60940-34-3, Ebselen 65005-57-4 73477-53-9 74209-34-0 148819-94-7 150403-89-7 156719-37-8 157254-42-7, Carbamimidothioic acid, 1,3-phenylenedi-2,1-ethanediyl ester 157254-60-9 159190-45-1 167423-51-0 209589-59-3, L-Ornithine, N5-[imino(methylthio)methyl]-, dihydrochloride 209913-88-2 211183-45-8 220805-22-1, L-Ornithine, N5-[(dimethylamino)iminomethyl]-, dihydrochloride
RL: COS (Cosmetic use); BIOL (Biological study); USES (Uses)
(use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin pigmentation disorders)

L161 ANSWER 5 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:695628 HCAPLUS
DN 137:221754
ED Entered STN: 13 Sep 2002
TI Use of subtances that prevent the effects of NO synthase in cosmetic preparations for the prophylaxis and treatment of skin inflammations and other disorders
IN Mummert, Christopher; Blatt, Thomas; Kolbe, Ludger; Wolber, Rainer; Kruse, Inge
PA Beiersdorf AG, Germany
SO Ger. Offen., 28 pp.

CODEN: GWXXBX
DT Patent
LA German
IC ICM A61K007-48
ICS A61K007-40
CC 62-3 (Essential Oils and Cosmetics)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 10111049	A1	20020912	DE 2001-10111049	20010306
PRAI	DE 2001-10111049		20010306		

AB The invention concerns substances that inhibit NO synthase activity or block the expression of NO synthase in warm blooded organisms and their application in cosmetic and dermatol. prepns. for the prevention and treatment of skin inflamation, atopic dermatitis and other disorders caused by sensitive and dry skin. Thus an O/W cream contained (weight/weight%): glycerylstearate 4.00; PEG-40-stearate 1.00; cetyl alc. 3.00; caprylic acid/caprylic acid triglyceride 5.00; mineral oil 5.00; nitroguanidine 0.10; tocopherol 0.10; trisodium EDTA 0.10; preservatives q.s.; carbomer 3.00; sodium hydroxide (45%) q.s.; glycerin 5.00; perfume q.s.; water to 100.
ST NO synthase inhibition skin cosmetics inflammation eczema
IT Dermatitis
(atopic; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin pigmentation disorders)
IT Cosmetics
(creams; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin inflammations and other disorders)
IT Cosmetics
(emulsions; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin inflammations and other disorders)
IT **Skin, disease**
(inflamation; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin inflammations and other disorders)
IT Ubiquinones
RL: COS (Cosmetic use); BIOL (Biological study); USES (Uses)
(use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin pigmentation disorders)
IT 125978-95-2, Synthase, **nitric oxide**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors of; use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin inflammations and other disorders)
IT 62-56-6, Thiourea, biological studies 123-78-4D, Sphingosine, N-Acyl derivs. 372-75-8, Citrulline 543-38-4, Canavanin 695-34-1, 2-Amino-4-Methylpyridine 2986-19-8, **S-Methylisothiourea** 3416-24-8, D-Glucosamine 60940-34-3, Ebselen
RL: COS (Cosmetic use); BIOL (Biological study); USES (Uses)
(use of subtances that prevent effects of NO synthase in cosmetic prepns. for prophylaxis and treatment of skin inflammations and other disorders)
IT 50-02-2, Dexamethasone 55-21-0, Benzamide 106-51-4, 2,5-Cyclohexadiene-1,4-dione, biological studies 143-37-3D, Acetamidine, fluorinated, amino acid and oleic acid derivs. 244-54-2, Dibenziodolium 244-63-3, Norharman 303-98-0, Coenzyme Q10 458-37-7 504-29-0,

2-Aminopyridine 556-88-7, Nitroguanidine 996-19-0 2149-70-4
2214-67-7D, Hetero derivs. 2582-07-2, (2-Benzothiazolyl)guanidine
2942-42-9 4269-97-0 4364-78-7, 1,3-Diaminoguanidine 4673-26-1
5154-02-9 17035-90-4 21463-31-0 22780-54-7, 2-Iminopiperidine
25371-96-4 31137-74-3 36889-13-1 39123-82-5 41137-86-4,
Hirsutanonol 41443-28-1 51298-62-5 55303-93-0, Oregonin 65005-57-4
73477-53-9 74209-34-0 148819-94-7 150403-89-7 156719-37-8
157254-42-7, Carbamimidothioic acid, 1,3-phenylenedi-2,1-ethanediyl ester
157254-60-9 159190-45-1 167423-51-0 209589-59-3, L-Ornithine,
N5-[imino(methylthio)methyl]-, dihydrochloride 209913-88-2 211183-45-8
220805-22-1, L-Ornithine, N5-[(dimethylamino)iminomethyl]-,
dihydrochloride
RL: COS (Cosmetic use); BIOL (Biological study); USES (Uses)
(use of subtances that prevent effects of NO synthase in cosmetic
prepns. for prophylaxis and treatment of skin pigmentation disorders)

RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

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(2) Anon; EP 0630649 A1 HCAPLUS
(3) Anon; DE 10000840 A1 HCAPLUS
(4) Anon; DE 19711565 A1 HCAPLUS
(5) Anon; DE 19918750 A1 HCAPLUS
(6) Anon; DE 19962267 A1 HCAPLUS
(7) Anon; FR 2744915 A1
(8) Anon; FR 2764504 A1 HCAPLUS
(9) Anon; DE 4341000 A1 HCAPLUS
(10) Anon; DE 4341001 A1 HCAPLUS
(11) Anon; US 5449688 A HCAPLUS
(12) Anon; WO 9513805 A1 HCAPLUS
(13) Anon; WO 9534534 A1 HCAPLUS
(14) Anon; WO 9626711 A1 HCAPLUS
(15) Anon; WO 9709032 A1 HCAPLUS
(16) Anon; WO 9709056 A1 HCAPLUS
(17) Anon; WO 9715280 A1 HCAPLUS
(18) Anon; WO 9809653 A1 HCAPLUS

L161 ANSWER 6 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:648411 HCAPLUS
DN 137:190741
ED Entered STN: 28 Aug 2002
TI **Nitrogen oxide** modulators for the treatment of **itching**
IN Kuraishi, Yasushi; Miyamoto, Takayuki
PA Ikeda Mohando Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 7 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
IC ICM A61K045-00
ICS A61K045-06; A61P017-04; A61P043-00; A61K031-223
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1

same application

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 2002241308	A2	20020828	JP 2001-44656	20010221
	US 2002156129	A1	20021024	US 2001-982380	20011017
PRAI	JP 2001-44656	A	20010221		

AB This invention relates to therapeutics for the treatment of itching which is caused by hypofunction of skin barriers. The agents for the treatment

of itching exhibit (1) NO biosynthesis-inhibiting activities and/or (2) NO-removing activities by binding with NO in vivo. For example, an ointment was formulated containing 5 % Nω-nitro-L-arginine Me ester.

ST **nitrogen oxide** inhibitor **itching** treatment; ointment **nitroarginine** methyl ester **itching**

IT **Pruritus**
(**nitrogen oxide** modulators for treatment of **itching**)

IT Hemoglobins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**nitrogen oxide** modulators for treatment of **itching**)

IT Drug delivery systems
(ointments; **nitrogen oxide** modulators for treatment of **itching**)

IT 125978-95-2, NO synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; **nitrogen oxide** modulators for treatment of **itching**)

IT 10102-43-9, Nitrogen monoxide, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**nitrogen oxide** modulators for treatment of **itching**)

IT **96-50-4**, **2-Aminothiazole 271-44-3**, **Indazole 2149-70-4**, **Nω - Nitro-L-arginine 2942-42-9**, **7-Nitroindazole 2986-19-8**, **S-Methylisothiourea 2986-20-1**, **S-Ethylisothiourea 6913-17-3 7597-18-4**, 6-Nitroindazole **13395-35-2**, 2-**Iminobiotin 17035-90-4**, **L-NMMA 25371-96-4 30315-93-6 36889-13-1** 50903-99-6, **L-NAME** 50961-01-8, **2-Aminothiazoline 53774-63-3 57444-72-1 139299-32-4 139461-37-3** 145157-47-7 **156719-37-8**, **L-Thiocitrulline 156719-38-9**, L-Homothiocitrulline **156719-41-4**, S-Methyl-**L-thiocitrulline 157254-42-7 158875-72-0**, **S-Ethyl-L-thiocitrulline 163490-54-8 168895-09-8**, AR-R17477 **180001-34-7** 451474-04-7, HMN 1180
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**nitrogen oxide** modulators for treatment of **itching**)

L161 ANSWER 7 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:615365 HCAPLUS
DN 137:159039
ED Entered STN: 16 Aug 2002
TI NOS inhibitors for treatment of wrinkles
IN Fujii, Seishiro; Lerner, Ethan
PA The General Hospital Corporation, USA
SO PCT Int. Appl., 15 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K007-00
ICS A61K007-42; A61K007-44; A61K031-495

CC 62-4 (Essential Oils and Cosmetics)
Section cross-reference(s): 63
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002062306	A1	20020815	WO 2002-US2292	20020125
	W: JP				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
	US 2002168325	A1	20021114	US 2002-57247	20020125
	EP 1359885	A1	20031112	EP 2002-720854	20020125
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
	US 2003207844	A1	20031106	US 2003-406306	20030403
PRAI	US 2001-264176P	P	20010125		
	US 2002-57247	A3	20020125		
	WO 2002-US2292	W	20020125		

AB Methods, compns., and kits, are provided for the use of nitric oxide synthase inhibitors to prevent and reduce wrinkles. L-NAME treated mice show a reduction in fine wrinkles compared to untreated control mice which can prevent the formation of wrinkles caused by UVB exposure.
ST NOS inhibitor wrinkle; LNAME wrinkle prevention
IT UV B radiation
(NOS inhibitors for treatment of wrinkles)
IT **Skin, disease**
(aging; NOS inhibitors for treatment of wrinkles)
IT Drug delivery systems
(topical; NOS inhibitors for treatment of wrinkles)
IT Cosmetics
(wrinkle-preventing; NOS inhibitors for treatment of wrinkles)
IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NOS inhibitors for treatment of wrinkles)
IT 50903-99-6, **L-Name**
RL: COS (Cosmetic use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NOS inhibitors for treatment of wrinkles)
RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Mjalli; US 5723451 A 1998 HCAPLUS

L161 ANSWER 8 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:579342 HCAPLUS
DN 138:130977
ED Entered STN: 05 Aug 2002
TI The effect of combined systemic administration of morphine and **L-NAME**, a **nitric oxide** synthase inhibitor, on behavioral signs of neuropathic pain in rats
AU Ulugol, Ahmet; Aslantas, Aysegul; Karadag, Hakan C.; Bulbul, Ebru D.; Tuncer, Alev; Dokmeci, Ismet
CS Department of Pharmacology, Faculty of Medicine, Trakya University, Edirne, 22030, Turk.
SO Neuroscience Research Communications (2002), 30(3), 143-153
CODEN: NRCOEE; ISSN: 0893-6609
PB Wiley-Liss, Inc.
DT Journal
LA English
CC 1-11 (Pharmacology)
AB The controversy over using opioids for managing chronic neuropathic pain is widely acknowledged, and NO is suggested to play an important role in

the maintenance of the behavioral signs of neuropathic pain. The authors evaluated the effect of combined systemic administration of morphine and NG-nitro-L-arginine-Me ester (L-NAME), a NO synthase (NOS) inhibitor, on allodynia in the spinal nerve ligation model of pain in rats. Nerve injury was produced by tight ligation of the left L5 and L6 spinal nerves and this procedure resulted in neuropathic pain behaviors in the ipsilateral hindlimb. Mech. and cold allodynia were detected, resp., by application of von Frey filaments or acetone to the plantar surface of the foot. Morphine (0.1-10 mg/kg, i.p.) and L-NAME (3-30 mg/kg, i.p.) reduced mech. and cold allodynia with their higher doses. Combining subthreshold dose of L-NAME (3 mg/kg, i.p.) with morphine, an appreciable increase in the antiallodynic effect of morphine was observed This effect was prevented by L-Arg (500 mg/kg, i.p.) and naloxone (1 mg/kg, i.p.). These results suggest that combining morphine with a NOS inhibitor may be a promising approach in the treatment of neuropathic pain.

ST morphine LNAME neuropathy allodynia analgesic

IT Pain
Skin, disease
(allodynia; combined systemic administration of morphine and **L-NAME** in neuropathic pain)

IT Analgesics
(combined systemic administration of morphine and **L-NAME** in neuropathic pain)

IT Nerve, disease
(neuropathy; combined systemic administration of morphine and **L-NAME** in neuropathic pain)

IT Nerve
(spinal; combined systemic administration of morphine and **L-NAME** in neuropathic pain)

IT 10102-43-9, **Nitric oxide**, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(combined systemic administration of morphine and **L-NAME** in neuropathic pain)

IT 57-27-2, Morphine, biological studies 50903-99-6, **L-NAME**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combined systemic administration of morphine and **L-NAME** in neuropathic pain)

RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

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L161 ANSWER 9 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:358835 HCAPLUS
DN 136:374800
ED Entered STN: 14 May 2002
TI Pharmaceuticals containing selective neuronal NO synthetase (NOS) inhibitors for treatment of **pruritis**
IN Wada, Yukihisa; Matsugu, Wataru; Yokota, Koichi
PA Japan Organo Co., Ltd., Japan; Human Science Shinko Zaidan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
IC ICM A61K045-00
ICS A61K031-155; A61K031-223; A61K031-381; A61P017-00; C07D333-38
CC 63-5 (Pharmaceuticals)
Section cross-reference(s): 1
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2002138052	A2	20020514	JP 2000-332335	20001031
PRAI JP 2000-332335		20001031		

AB Title pharmaceuticals are claimed. Thus, AR-R17477 inhibited neuronal, endothelial, and inducible NOS with IC50 of 1.8, 81, and 28 μM, resp., and at 0.1-10 mg/kg i.v. strongly inhibited itching in atopic dermatitis

model mice without causing hypertension.
ST atopic dermatitis **pruritis** treatment **ARR17477**; **nitric oxide** synthetase inhibitor treatment **pruritis**
IT Dermatitis
(atopic; selective neuronal NO synthetase inhibitors for treatment of **pruritis**)
IT **Pruritus**
(selective neuronal NO synthetase inhibitors for treatment of **pruritis**)
IT 125978-95-2, **Nitric oxide** synthetase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(neuronal; selective neuronal NO synthetase inhibitors for treatment of **pruritis**)
IT **168895-09-8**, AR-R17477 180001-47-2
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(selective neuronal NO synthetase inhibitors for treatment of **pruritis**)

L161 ANSWER 10 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:122326 HCAPLUS
DN 137:304459
ED Entered STN: 15 Feb 2002
TI Therapeutic administration of **nitric oxide** synthase inhibitors reverses hyperalgesia but not inflammation in a rat model of polyarthritis
AU Tedesco, Laura S.; Fuseler, John; Grisham, Matthew; Wolf, Robert; Roerig, Sandra C.
CS Department of Pharmacology and Therapeutics, Louisiana State University Health Sciences Center, Shreveport, LA, 71130, USA
SO Pain (2002), 95(3), 215-223
CODEN: PAINDB; ISSN: 0304-3959
PB Elsevier Science B.V.
DT Journal
LA English
CC 1-7 (Pharmacology)
AB Nitric oxide (NO) has been postulated to play a role in pain as well as in inflammation. In the present studies, the effects of NO synthase (NOS) inhibitors on both pain and inflammation were examined in a rat model of polyarthritis. Female Lewis rats were injected i.p. with peptidoglycan/polysaccharide (PG/PS) or saline to induce arthritis. Hind paw volume, response latency to thermal nociceptive stimulus and mech. threshold were measured daily for the next 35 days. Paw inflammation, thermal hyperalgesia and mech. allodynia developed in all rats that received PG/PS compared to saline. On day 19 (chronic inflammation phase), rats were given either NG-nitro-l-arginine Me ester (l-NAME, non-selective NOS inhibitor, 100 mg/l), l-N (6)-(1-iminoethyl) lysine (l-NIL, selective inducible NOS inhibitor, 10 mg/l) or no drug in drinking water. By day 21, l-NAME treatment reversed the thermal hyperalgesia completely and this effect remained until day 35. Similarly, l-NIL treatment reversed thermal hyperalgesia from days 24 to 34. Neither treatment affected mech. allodynia. Paw volume was not different between PG/PS treated and PG/PS plus l-NAME treated rats. However, the PG/PS plus l-NIL treatment produced an increase in paw volume greater than did PG/PS alone. Other rats were treated with PG/PS plus the antiinflammatory agent indomethacin (days 19-35). Indomethacin treatment reversed all the measured parameters, although the reversal of mech. allodynia was only partial. These results suggest that NO is involved in thermal, but not mech. sensory pathways and that the selective inhibition of inducible NOS

activity exacerbates established inflammation.
ST NO synthase inhibitor hyperalgesia inflammation polyarthritis
IT Analgesics
Anti-inflammatory agents
Antiarthritics
Inflammation
(NOS inhibitors reverse hyperalgesia but not inflammation in polyarthritis)
IT Pain
Skin, disease
(allodynia; NOS inhibitors reverse hyperalgesia but not inflammation in polyarthritis)
IT Pain
(hyperalgesia; NOS inhibitors reverse hyperalgesia but not inflammation in polyarthritis)
IT Arthritis
(polyarthritis; NOS inhibitors reverse hyperalgesia but not inflammation in polyarthritis)
IT 53-86-1, Indomethacin 50903-99-6, **L-NAME**
53774-63-3
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NOS inhibitors reverse hyperalgesia but not inflammation in polyarthritis)
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
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L161 ANSWER 11 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:851123 HCAPLUS
DN 136:5985
ED Entered STN: 23 Nov 2001
TI Preparation of tricyclic pyrazole derivatives as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases
IN Doyle, Kevin J.; Rafferty, Paul; Steele, Robert W.; Wilkins, David J.; Arnold, Lee D.; Hockley, Michael; Ericsson, Anna M.; Iwasaki, Nobuhiko; Ogawa, Nobuo
PA Knoll G.m.b.H., Germany
SO PCT Int. Appl., 183 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07D231-00
CC 28-8 (Heterocyclic Compounds (More Than One Hetero Atom))
Section cross-reference(s): 1
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001087846	A2	20011122	WO 2001-US16153	20010517
	WO 2001087846	A3	20020321		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6462036	B1	20021008	US 2000-573366	20000517
	EP 1289525	A2	20030312	EP 2001-937553	20010517
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2003533514	T2	20031111	JP 2001-584242	20010517
PRAI	US 2000-573366	A1	20000517		
	US 1998-107467P	P	19981106		
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OS MARPAT 136:5985

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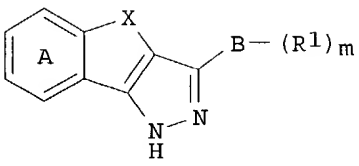

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AB Title compds. I [m = 1-10; X = (CH2)n, CO, O, C:NOR10, NR11, (CH2)n, S, SO, or SO2; n = 1-3; R10 = alkyl; R11 = (un)substituted alkyl or Ph; B = (cyclo)alkyl, aryl, pyridyl, thienyl, furyl, or pyrrolyl; R1 = H, halo, OH, NO2, CN, hydroxyamidino, CH2NH2, formamidomethyl, (un)substituted alkenyl(oxy), alkynyl, or YW; Y = absent or alkyl, alkoxy, O, S, or CO; W = H, OH, (un)substituted Ph, alkoxy, or amino; ring A is optionally substituted with halo, OH, NO2, CN, or (un)substituted alkyl, alkoxy, PhO, carboxy, carbamoyl, amino, amido, aralkyl, alkenyl, or alkynyl; with provisos; and racemic mixts., racemic diastereomeric mixts., tautomers, optical isomers, and pharmaceutically acceptable salts thereof] were prepared as protein kinase inhibitors, especially tyrosine kinase inhibitors. Thus, indan-1-one hydrazone (preparation given) in THF at 0° was treated with BuLi and then with Me 3,4,5-trimethoxybenzoate to give 3-(3,4,5-trimethoxyphenyl)-1,4-dihydroindeno[1,2-c]pyrazole. Example compds. significantly inhibited KDR kinase at concns. of ≤ 50 μM.

ST tricyclic pyrazole prepn tyrosine kinase inhibitor; pyrazole tricyclic prepn vascular hyperpermeability inhibitor; angiogenesis inhibitor tricyclic pyrazole prepn; indenopyrazole prepn protein kinase inhibitor

IT Respiratory distress syndrome
(adult, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Antiarteriosclerotics
(antiatherosclerotics; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Fever and Hyperthermia
(cat **scratch**; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related **diseases**)

IT Lung, disease
(chronic, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Eye, disease
(diabetic retinopathy, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Helicobacter
(disease treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Brain, disease
Lung, disease
(edema, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Body fluid
(effusion, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Uterus, disease
(endometriosis, treatment; preparation of tricyclic pyrazole derivs. as

tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Bone, disease
(fracture, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Antitumor agents
Blood vessel, neoplasm
(hemangioma; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Ovary, disease
(hyperstimulation syndrome, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Eye, disease
(keratopathy, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Eye, disease
(macula, degeneration, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Nerve, disease
(peripheral, injury, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Nose, neoplasm
(polyp, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Angiogenesis inhibitors
Anti-inflammatory agents
Antiarthritics
Antiglaucoma agents
Antitumor agents
Antiulcer agents
Wound healing promoters
(preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Eye, disease
(retinopathy, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Brain, disease
(stroke, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Synovial membrane, disease
(synovitis, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Hyperplasia
(thyroid; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Injury
(trauma, treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT Ascites
Blood vessel, disease
Burn
Cirrhosis
Cyst, pathological
Fibrosis
Keloid

Multiple organ failure
Psoriasis
Sepsis
(treatment; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 80449-02-1, Tyrosine kinase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibitors; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 5736-44-7P, Indan-1-one hydrazone 29045-95-2P, Methyl 4-(1-oxoindan-2-ylidenemethyl)benzoate 222965-05-1P, 3-(3-Methoxyphenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268563-20-8P, Methyl 4-[1-oxospiro[indan-2,2'-oxiran]-3'-yl]benzoate 268563-21-9P, N-(2-Benzylidene-1-oxoindan-5-yl)acetamide 268563-22-0P, N-[1-Oxo-3'-phenylspiro[indan-2,2'-oxiran]-5-yl]acetamide 268563-23-1P, 4'-(1-Benzoyl-1,4-dihydroindeno[1,2-c]pyrazol-3-yl)benzanilide 268563-24-2P, 2-[4-Hydroxy-3-(hydroxymethyl)benzylidene]-1-indanone 268563-87-7P, 4-[1-Oxospiro[indan-2,2'-oxiran]-3'-yl]benzoic acid 268564-01-8P, N-(1-Chloroacetyl-3-phenyl-1,4-dihydroindeno[1,2-c]pyrazol-6-yl)-2-chloroacetamide 374903-21-6P, N-[4-(1-Benzylsulfonyl-1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]benzenesulfonamide 374903-22-7P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(intermediate; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 268559-60-0P, 3-(1,4-Dihydroindeno-[1,2-c]pyrazol-3-yl)phenol 268559-61-1P, 3-Phenyl-1H-[1]benzothieno[3,2-c]pyrazole 268559-72-4P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)benzoic acid 268559-73-5P, Methyl 4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)benzoate 268559-77-9P, 4'-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)morpholinoacetanilide 268560-07-2P, 3-(4-Nitrophenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268560-08-3P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)aniline 268561-96-2P, N-(3-Phenyl-1,4-Dihydroindeno[1,2-c]pyrazol-6-yl)-2-morpholinoacetamide 268562-37-4P, 3-(4-Bromophenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268562-48-7P, 3-(4-Bromophenyl)-1H-[1]benzothieno[3,2-c]pyrazole 268563-28-6P, 4-[1H-[1]Benzothieno[3,2-c]pyrazol-3-yl]benzaldehyde
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 54030-69-2P 268559-59-7P, 3-(3,4,5-Trimethoxyphenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268559-62-2P, 3-(2-Thienyl)-1H-benzothieno[3,2-c]pyrazole 268559-63-3P, 3-Phenyl-1H-[1]benzothieno[3,2-c]pyrazole 4-oxide 268559-64-4P, 3-Phenylindeno[1,2-c]pyrazol-4(1H)-one oxime 268559-78-0P, 4'-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)benzanilide 268559-79-1P, N-(3-Phenyl-1,4-dihydroindeno[1,2-c]pyrazol-6-yl)acetamide 268559-81-5P, N-(3-Phenyl-1,4-dihydroindeno[1,2-c]pyrazol-6-yl)benzamide 268559-82-6P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)benzamide 268559-83-7P, N-Methyl-4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)benzamide 268559-84-8P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)benzanilide 268559-85-9P, N-(2-Diethylaminoethyl)-4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)benzamide 268559-86-0P, N-(2-Morpholinoethyl)-4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)benzamide 268559-87-1P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenol 268559-89-3P, 3-(2-Thienyl)-1,4-dihydroindeno[1,2-c]pyrazol-6-ol 268559-91-7P, 2-[3-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenoxy]ethanol 268559-92-8P,

3-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenoxyacetic acid 268559-93-9P, Ethyl 3-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenoxyacetate 268559-94-0P 268559-97-3P, 4-[3-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenoxy]butyric acid 268559-98-4P, Ethyl 4-[3-(1,4-dihydroindeno-[1,2-c]pyrazol-3-yl)phenoxy]butyrate 268559-99-5P, 4-[3-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenoxy]butyramide 268560-09-4P, 4-(4,5-Dihydro-1H-benzo[g]indazol-3-yl)pyridine 1-oxide 268560-16-3P, 2-Morpholinoethyl 4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)benzoate 268560-17-4P, 3-(3-Nitrophenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268560-18-5P, 3-(4-Methylthiophenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268560-20-9P, 3-(2-Naphthyl)-1,4-dihydroindeno[1,2-c]pyrazole 268560-22-1P, 3-(4-Difluoromethoxyphenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268560-24-3P, 3-(4-Acetamidophenyl)-4,5-dihydro-2H-benz[g]indazole 268560-26-5P, 3-(4-Bromo-2-thienyl)-1,4-dihydroindeno[1,2-c]pyrazole 268560-28-7P, 3-(4-Benzyloxyphenyl)-4,5-dihydro-2H-benz[g]indazole 268560-30-1P, 6,7-Dimethoxy-3-(3-phenoxyphenyl)-1,4-dihydroindeno-[1,2-c]pyrazole 268560-31-2P, 3-[4-(5-Trifluoromethyl-2-pyridyloxy)phenyl]-1,4-dihydroindeno[1,2-c]pyrazole 268560-32-3P, 6,7,8-Trimethoxy-3-(2,3,4-trimethoxyphenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268560-33-4P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)-2-(hydroxymethyl)phenol 268560-34-5P, 2-Methoxy-5-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenol 268560-35-6P, 2-Chloro-4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenol 268560-36-7P, 2-Methoxy-4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenol 268560-37-8P, 3-Chloro-4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenol 268560-38-9P, 2-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenoxy]acetamide 268560-39-0P, 4'-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)diethylaminoacetanilide 268560-40-3P, 4-[1H-[1]Benzothieno[3,2-c]pyrazol-3-yl]benzamide 268560-41-4P, 3-(4-Aminophenyl)-1H-[1]benzothieno[3,2-c]pyrazole 268560-42-5P, 3-(4-Methoxyphenyl)-1H-benzothieno[3,2-c]pyrazole 268560-43-6P, 3-(4-Hydroxyphenyl)-1H-[1]-benzothieno[3,2-c]pyrazole 268560-44-7P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)benzonitrile 268560-45-8P 268560-46-9P, 4-Methyl-3-phenyl-1,4-dihydroindeno[1,2-c]pyrazol-4-ol 268560-50-5P 268561-53-1P 268561-57-5P 268561-95-1P 268562-05-6P 268562-23-8P 268562-24-9P 268562-25-0P 268562-26-1P 268562-27-2P 268562-29-4P 268562-31-8P 268562-33-0P 268562-39-6P 268562-42-1P 268562-45-4P 268562-50-1P 268562-52-3P 268562-54-5P 268562-56-7P 268562-58-9P 268562-60-3P 268562-62-5P 268562-64-7P 268562-67-0P 268562-68-1P 268562-69-2P 268562-70-5P 268562-71-6P 268562-72-7P 268562-73-8P 268562-74-9P 268562-75-0P 268562-76-1P 268562-77-2P 268562-78-3P 268562-79-4P 268562-80-7P 268562-81-8P 268562-82-9P 268562-83-0P 268562-84-1P 268562-85-2P 268562-86-3P 268562-87-4P 268562-88-5P 268562-89-6P 268562-90-9P 268562-91-0P 268562-92-1P 268562-93-2P 268562-94-3P 268562-95-4P 268562-96-5P 268562-97-6P 268562-98-7P 268562-99-8P 268563-00-4P 268563-01-5P 268563-02-6P 268563-03-7P 268563-04-8P 268563-05-9P 268563-06-0P 268563-07-1P 268563-08-2P 268563-09-3P 268563-10-6P 268563-11-7P 268563-12-8P 268563-13-9P 268563-14-0P 268563-15-1P 268563-16-2P 268563-17-3P 268563-18-4P 268563-19-5P 268563-27-5P 268563-29-7P, 4-[1H-[1]Benzothieno[3,2-c]pyrazol-3-yl]-N-[3-(imidazol-1-yl)propyl]benzylamine trihydrochloride 268563-30-0P, Methyl 4-(4-oxo-1,4-dihydroindeno[1,2-c]pyrazol-3-yl)benzoate 268563-32-2P 268563-33-3P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]benzenesulfonamide 268563-34-4P 268563-36-6P, N-(2-Morpholinoethyl)-3-phenyl-1,4-dihydroindeno[1,2-c]pyrazol-6-ylamine trihydrochloride 268563-37-7P, 3-[4-(2-Morpholinoethoxy)phenyl]-1,4-dihydroindeno[1,2-c]pyrazole 268563-38-8P, 3-[2-(2H-1,2,3,4-Tetraazol-5-yl)-4-pyridyl]-4,5-dihydro-2H-benzo[g]indazole 268563-39-9P, 3-(4-Isocyanatophenyl)-1,4-dihydroindeno[1,2-c]pyrazole 268563-40-2P, 2-(Diethylamino)ethyl N-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]carbamate 268563-41-3P, 2-Morpholinoethyl N-[4-[1,4-

dihydroindeno[1,2-c]pyrazol-3-yl]phenyl]carbamate 268563-42-4P 268563-45-7P, 1-Methyl-2-propoxyethyl N-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]carbamate 268563-46-8P, 2-(1-Methyltetrahydro-1H-2-pyrrolyl)ethyl N-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]carbamate 268563-49-1P, N-[2-(Diethylamino)ethyl]-N'-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]urea 268563-50-4P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-(2-morpholinoethyl)urea 268563-51-5P, N1-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-1-piperidinecarboxamide 268563-52-6P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N-[2-(dimethylamino)-1-methylethyl]urea 268563-53-7P 268563-54-8P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N1-(2-furylmethyl)urea 268563-55-9P, N-(1,3-Benzodioxol-5-ylmethyl)-N'[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]urea 268563-56-0P, N-Cyclobutyl-N'-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]urea 268563-57-1P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-(2-piperidinoethyl)urea 268563-58-2P, N-Benzyl-N'-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]urea 268563-59-3P, N-[4-(Diethylamino)butyl]-N'-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]urea 268563-60-6P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-[2-(2-thienyl)ethyl]urea 268563-61-7P, N-[3-(Diethylamino)propyl]-N'-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]urea 268563-62-8P, N-[4-[1,4-Dihydroindeno[1,2-c]pyrazol-3-yl]phenyl]-N'-[(1-ethyltetrahydro-1H-2-pyrrolyl)methyl]urea 268563-63-9P, N-(2,5-Difluorobenzyl)-N'-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]urea 268563-64-0P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-[2-(2-hydroxyethoxy)ethyl]urea 268563-65-1P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-[2-hydroxy-1-(hydroxymethyl)ethyl]urea 268563-66-2P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-(2,3-dihydroxypropyl)urea 268563-67-3P, N1-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-4-(2-pyridyl)-1-piperazinecarboxamide 268563-68-4P, N'-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N-[3-(dimethylamino)propyl]-N-methylurea 268563-69-5P 268563-70-8P 268563-71-9P, N-Benzyl-N'-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N-methylurea 268563-72-0P, N'-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N-ethyl-N-(2-hydroxyethyl)urea 268563-73-1P 268563-74-2P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N-[2-(dimethylamino)ethyl]-N-methylurea 268563-75-3P, N1-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-4-methyl-1-piperazinecarboxamide 268563-76-4P 268563-77-5P 268563-78-6P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-4-phenyl-1-piperazinecarboxamide 268563-79-7P, N'-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N,N-di(2-methoxyethyl)urea 268563-80-0P, N'-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N-(2,3-dihydroxypropyl)-N-methylurea 268563-81-1P, N,N-Di[2-(Diethylamino)ethyl]-N'-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]urea 268563-82-2P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-(2-pyridylmethyl)urea 268563-83-3P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-(3-pyridylmethyl)urea 268563-84-4P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-(4-pyridylmethyl)urea 268563-85-5P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-(2-hydroxyethyl)urea 268563-86-6P, N-[4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]-N'-[7-(dimethylamino)heptyl]urea 268564-03-0P, N-(2-Diethylaminoethyl)-4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)benzamide dihydrochloride 268564-04-1P, 4'-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)acetanilide dihydrochloride 268725-18-4P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)phenol sodium salt 362611-30-1P 374903-14-7P, 3-Phenyl-1,4-dihydroindeno[1,2-c]pyrazol-6-ylamine 374903-20-5P, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)benzamide oxime 374903-23-8P 374903-24-9P 374903-25-0P, 2-[Ethyl(2-hydroxyethyl)amino]ethyl N-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-

yl)phenyl]carbamate acetate 374903-26-1P, 2-[[2-(Dimethylamino)ethyl](methyl)amino]ethyl N-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]carbamate acetate 374903-27-2P, 2-[2-(Dimethylamino)ethoxy]ethyl N-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]carbamate acetate 374903-28-3P, 2-(Diethylamino)-1-methylethyl N-[4-(1,4-dihydroindeno[1,2-c]pyrazol-3-yl)phenyl]carbamate acetate
374903-31-8P 374903-32-9P 374903-33-0P 374903-34-1P 374903-35-2P
374903-36-3P 374903-37-4P 374903-38-5P 374903-39-6P 374903-40-9P
374903-41-0P 374903-42-1P 374903-43-2P 374903-44-3P 374903-45-4P
374903-46-5P 374903-47-6P 374903-48-7P 374903-49-8P 374903-50-1P
374903-52-3P 374903-53-4P 374903-54-5P 374903-55-6P 374903-56-7P
374903-57-8P 374903-58-9P 374903-59-0P 374903-60-3P 374903-61-4P
374903-62-5P 374903-63-6P 374903-64-7P 374903-65-8P 374903-66-9P
374903-67-0P 374903-68-1P 374903-69-2P 374903-70-5P 374903-71-6P
374903-72-7P 374903-73-8P 374903-74-9P 374903-75-0P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 374903-76-1P 374903-77-2P 374903-78-3P 374903-79-4P 374903-80-7P
374903-81-8P 374903-82-9P 374903-83-0P 374903-84-1P 374903-85-2P
374903-86-3P 374903-87-4P 374903-88-5P 374903-89-6P 374903-90-9P
374903-91-0P 374903-92-1P 374903-93-2P 374903-94-3P 374903-95-4P
374903-96-5P 374903-97-6P 374903-98-7P 374903-99-8P 374904-00-4P
374904-01-5P 374904-02-6P 374904-03-7P 374904-04-8P 374904-05-9P
374904-06-0P 374904-07-1P 374904-08-2P 374904-09-3P 374904-10-6P
374904-11-7P 374904-12-8P 374904-13-9P 374904-14-0P 374904-15-1P
374904-16-2P 374904-17-3P 374904-18-4P 374904-19-5P 374904-20-8P
374904-21-9P 374904-22-0P 374904-23-1P 374904-24-2P 374904-25-3P
374904-26-4P 374904-27-5P 374904-28-6P 374904-29-7P 374904-30-0P
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374904-36-6P 374904-37-7P 374904-38-8P 374904-39-9P 374904-40-2P
374904-41-3P 374904-42-4P 374904-43-5P 374904-44-6P 374904-45-7P
374904-46-8P 374904-47-9P 374904-48-0P 374904-49-1P 374904-50-4P
374904-51-5P 374904-52-6P 374904-53-7P 374904-54-8P 374904-55-9P
374904-56-0P 374904-57-1P 374904-58-2P 374904-59-3P 374904-60-6P
374904-61-7P 374904-62-8P 374904-63-9P 374904-64-0P 374904-65-1P
374925-18-5P 374925-19-6P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 114051-78-4, Lck kinase 141349-87-3, Fyn kinase 141349-89-5, Src kinase 141349-91-9, Yes protein kinase 141350-03-0, Flt-1 kinase 148047-29-4, TIE-2 kinase 150977-45-0, Gene KDR protein kinase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 374903-18-1P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 374903-19-2P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

IT 61-82-5, 1H-1,2,4-Triazol-3-amine 75-31-0, 2-Propanamine, reactions 83-33-0, Indan-1-one **96-50-4**, 2-Thiazolamine 98-09-9, Benzenesulfonyl chloride 99-98-9 100-36-7, N,N-Diethylethylenediamine 100-37-8, N,N-Diethyl ethanol amine 104-94-9 105-07-7, 4-Cyanobenzaldehyde 105-36-2, Ethyl 2-bromoacetate 107-11-9, 2-Propen-1-amine 108-00-9 108-91-8, Cyclohexanamine, reactions 109-01-3 109-85-3 110-89-4, Piperidine, reactions 110-91-8, Morpholine, reactions 111-26-2, 1-Hexanamine 121-33-5, 4-Hydroxy-3-methoxybenzaldehyde 123-30-8 124-22-1, 1-Dodecanamine 462-08-8, 3-Pyridinamine 504-29-0, 2-Pyridinamine 621-59-0, 3-Hydroxy-4-methoxybenzaldehyde 622-40-2, 2-Morpholinoethanol 683-57-8, 2-Bromoacetamide 765-30-0, Cyclopropanamine 1571-08-0, Methyl 4-formylbenzoate 1916-07-0, Methyl 3,4,5-trimethoxybenzoate 2038-03-1, N-(2-Aminoethyl)morpholine 2420-16-8, 3-Chloro-4-hydroxybenzaldehyde 2450-71-7, 2-Propyn-1-amine 2969-81-5, Ethyl 4-bromobutyrate 3399-73-3, 1-Cyclohexene-1-ethanamine 3731-51-9, 2-Pyridinemethanamine 3731-52-0, 3-Pyridinemethanamine 3731-53-1, 4-Pyridinemethanamine 4418-61-5, 1H-Tetrazol-5-amine 5036-48-6, 3-(Imidazol-1-yl)propylamine 5049-61-6, Pyrazinamine 7144-05-0, 4-Piperidinemethanamine 7720-39-0, 1H-Imidazol-2-amine 10259-22-0, Ethyl 3-methoxybenzoate 15836-30-3, 2-(4-Methoxybenzoyl)-3-hydroxybenzo[b]thiophene 27578-60-5, 1-Piperidineethanamine 54030-32-9, 4-Hydroxy-3-(hydroxymethyl)benzaldehyde 55405-42-0, 2-Benzoylbenzo[b]thiophen-3(2H)-one 56767-20-5, 3-Phenylindeno[1,2-c]pyrazol-4(1H)-one 56962-11-9, 2-Chloro-4-hydroxybenzaldehyde 58161-35-6, N-(1-Oxoindan-5-yl)acetamide 58585-12-9, 2-(4-Nitrobenzoyl)-3-hydroxybenzo[b]thiophene 62985-37-9, 4-Morpholinemethanamine 68063-19-4, 2-(3-Nitrobenzylidene)-1-indanone 92882-97-8, 2-(2-Naphthylmethylene)-1-indanone 92882-99-0, 4-(1-Oxoindan-2-ylidenemethyl)phenol 154226-83-2, 2-(4-Benzyloxybenzylidene)-1-tetralone 194722-73-1, 2-(4-Acetamidobenzylidene)-1-tetralone 257284-87-0, 5,6,7-Trimethoxy-2-(2,3,4-trimethoxybenzylidene)-1-indanone 268563-88-8, 3'-(4-Nitrophenyl)-1-oxospiro[indan-2,2'-oxirane] 268563-89-9 268563-90-2, 4-(1,4-Dihydroindeno[1,2-c]pyrazol-3-yl)benzoyl chloride 268563-91-3, 2-(4-Methylthiobenzylidene)-1-indanone 268563-92-4, 2-(4-Difluoromethoxybenzylidene)-1-indanone 268563-93-5, 2-(4-Bromo-2-thienylmethylene)-1-indanone 268563-94-6, 5,6-Dimethoxy-2-(3-phenoxybenzylidene)-1-indanone 268563-95-7, 2-[4-(5-Trifluoromethyl-2-pyridyloxy)benzylidene]-1-indanone 268563-97-9, 4'-(1-Chloroacetyl-1,4-dihydroindeno[1,2-c]pyrazol-3-yl)chloroacetanilide 268563-98-0, 2-(4-Cyanobenzoyl)-3-hydroxybenzo[b]thiophene 268563-99-1, N-(2-Benzylidene-1-oxoindan-5-yl)benzamide 268564-00-7, 6-Methoxy-2-(4-methylthiobenzylidene)-1-tetralone 268564-02-9 328063-34-9 374903-15-8 374903-16-9 374903-17-0, 6-Methoxy-1-tert-butyloxycarbonyl-3-(2-thienyl)-1,4-dihydroindeno[1,2-c]pyrazole 374903-30-7 374904-66-2
RL: RCT (Reactant); RACT (Reactant or reagent)
(reactant; preparation of tricyclic pyrazole derivs. as tyrosine kinase inhibitors for treatment of angiogenesis-related diseases)

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ED Entered STN: 19 Jul 2001
TI Use of NO synthase inhibitors in skin preparations
IN Schoenrock, Uwe; Hinze, Claudia; Wilke, Jochen; Kruse, Inge
PA Beiersdorf A.-G., Germany
SO Ger. Offen., 8 pp.
CODEN: GWXXBX

DT Patent
LA German
IC ICM A61K007-48
CC 62-4 (Essential Oils and Cosmetics)
Section cross-reference(s): 63
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 10000840	A1	20010719	DE 2000-10000840	20000112
PRAI DE 2000-10000840		20000112		

AB The invention concerns the application of the NO synthase inhibitor nitroarginine in skin formulations to prevent aging of the skin. Thus a O/W cream contained in weight/weight%: glyceryl stearate 5.00; cetyl alc. 5.00; isopropylpalmitate 7.00; cyclomethicone 0.10; nitroarginine 0.10; NaOH (40%) 1.00; butylene glycol 3.00; Na2H2EDTA 0.20; ethanol 2.00; perfume, stabilizers, dyes q.s.; water to 100.
ST NO synthase inhibitor **nitroarginine** skin prepn aging
IT **Skin, disease**
(aging; use of NO synthase inhibitors in skin prepns.)
IT Cosmetics
(creams; use of NO synthase inhibitors in skin prepns.)
IT Cosmetics
(gels; use of NO synthase inhibitors in skin prepns.)
IT Cosmetics
(lotions; use of NO synthase inhibitors in skin prepns.)
IT Drug delivery systems
(ointments, creams; use of NO synthase inhibitors in skin prepns.)
IT Skin preparations (pharmaceutical)
(use of NO synthase inhibitors in skin prepns.)
IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; use of NO synthase inhibitors in skin prepns.)
IT 2149-70-4, **Nitroarginine**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(use of NO synthase inhibitors in skin prepns.)
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Anon; WO 9626711 A1 HCAPLUS
(2) Anon; WO 9715280 A1 HCAPLUS
(3) Anon; WO 9809653 A1 HCAPLUS

L161 ANSWER 13 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:95184 HCAPLUS
DN 134:275691
ED Entered STN: 08 Feb 2001
TI A murine model of opioid-induced hyperalgesia
AU Li, X.; Angst, M. S.; Clark, J. D.
CS Department of Anesthesiology, Veterans Affairs Palo Alto Health Care System and Stanford University, Palo Alto, CA, 94304, USA
SO Molecular Brain Research (2001), 86(1,2), 56-62
CODEN: MBREE4; ISSN: 0169-328X
PB Elsevier Science B.V.
DT Journal
LA English
CC 1-11 (Pharmacology)
Section cross-reference(s): 14
AB Controversies surround the possible long-term physiol. and psychol. consequences of opioid use. Analgesic tolerance and addiction are commonly at the center of these controversies, but other concerns exist as

well. A growing body of evidence suggests that hyperalgesia caused by the chronic administration of opioids can occur in laboratory animals and in humans.

In these studies we describe a murine model of opioid-induced hyperalgesia (OIH). After the treatment of mice for 6 days with implanted morphine pellets followed by their removal, both thermal hyperalgesia and mech. allodynia were documented. Addnl. expts. demonstrated that prior morphine treatment also increased formalin-induced licking behavior. These effects were intensified by intermittent abstinence accomplished through administration of naloxone during morphine treatment. Expts. designed to determine if the μ-opioid receptor mediated OLH in our model revealed that the relatively-selective μ-opioid receptor agonist fentanyl induced the thermal hyperalgesia and mech. allodynia characteristic of OIH when administered in intermittent boluses over 6 days. In complimentary expts. we found that CXBK mice which have reduced μ-opioid receptor binding displayed no significant OIH after morphine treatment. Finally, we explored the pharmacol. sensitivities of OIH. We found that the N-methyl-d-aspartate (NMDA) receptor antagonist MK-801, the nitric oxide synthase (NOS) inhibitor NG-nitro-l-arginine Me ester (l-NAME) and the heme oxygenase (HO) inhibitor tin protoporphyrin (Sn-P) dose-dependently reduced OIH in this model while the NSAID indomethacin had no effect. Thus we have characterized a murine model of OIH which will be useful in the pursuit of the mol. mechanisms underlying this phenomenon.

ST murine model opioid hyperalgesia mol mechanism

IT Glutamate receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NMDA-binding; murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT Pain
Skin, disease
(allodynia; murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT Pain
(hyperalgesia; murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT Behavior
(licking; murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT Disease models
Drug withdrawal
(murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT Opioids
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT Opioid receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(μ-opioid; murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT 57-27-2, Morphine, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT 53-86-1, Indomethacin 14325-05-4, Tin protoporphyrin 50903-99-6, **l-NAME** 77086-22-7, MK-801
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)

IT 9059-22-7, Heme oxygenase 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(murine model of opioid-induced hyperalgesia: mol. mechanisms underlying this phenomenon)
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
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(2) Ali, N; Anesthesiology 1986, V65, P449 MEDLINE
(3) Celerier, E; Anesthesiology 2000, V92, P465 HCAPLUS
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(6) Clark, J; Neurosci Lett 1998, V251, P121 HCAPLUS
(7) Dalman, F; J Neurosci 1999, V19, P5750 HCAPLUS
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(23) Moskowitz, A; Brain Res 1985, V361, P46 HCAPLUS
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(29) Solomon, R; Psychol Rev 1974, V81, P119 MEDLINE
(30) Suzuld, T; Pharmacol Biochem Behav 1997, V57, P293
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L161 ANSWER 14 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:585958 HCAPLUS
DN 129:207217
ED Entered STN: 15 Sep 1998
TI **Nitric oxide** synthase inhibitors for treatment of rosacea
IN Sauermann, Gerhard; Hoppe, Udo; Diembeck, Walter; Steinkraus, Volker; Salzer, Birgit
PA Beiersdorf A.-G., Germany
SO Ger. Offen., 10 pp.
CODEN: GWXXBX
DT Patent
LA German
IC ICM A61K031-195
ICS A61K031-17
ICI A61K031-195, A61K031-17
CC 63-6 (Pharmaceuticals)
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI DE 19711565 A1 19980827 DE 1997-19711565 19970320
WO 9836730 A2 19980827 WO 1998-EP991 19980220
WO 9836730 A3 19981022
W: JP, US
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
EP 969790 A2 20000112 EP 1998-913571 19980220
R: AT, BE, CH, DE, ES, FR, GB, IT, LI, NL, SE
JP 2001512471 T2 20010821 JP 1998-536269 19980220
PRAI DE 1997-19706581 A1 19970221
DE 1997-19711565 A 19970320
WO 1998-EP991 W 19980220
AB Rosacea and cuperosis may be prevented and/or treated, especially topically, with NO synthase inhibitors and their derivs. Cosmetic or dermatol. prepns. containing these agents may addnl. contain antioxidants, UV filters, and/or inorg. pigments. Thus, a sunscreen gel contained NG-nitro-L-arginine Me ester-HCl 1, benzophenone-4 0.5, phenylbenzimidazolesulfonic acid 1.3, acrylamide/Na acrylate copolymer 1.6, EtOH 5.0, glycerin 15.0, 15% NaOH for pH adjustment, perfume, preservative, and deionized water to 100.0 weight%.
ST rosacea **nitric oxide** synthase inhibitor
IT **Skin, disease**
(rosacea; **nitric oxide** synthase inhibitors for treatment of rosacea)
IT Drug delivery systems
(topical; **nitric oxide** synthase inhibitors for treatment of rosacea)
IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; **nitric oxide** synthase inhibitors for treatment of rosacea)
IT 867-44-7 2149-70-4 2942-42-9, **7-Nitroindazole**
13395-35-2, 2-**Iminobiotin** 36889-13-1 40911-12-4 50903-99-6
51298-62-5 53308-83-1 53774-63-3 150403-88-6 156719-41-4,
S-Methyl-**L-thiocitrulline** 159190-45-1 200716-66-1
209248-80-6 212051-53-1
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**nitric oxide** synthase inhibitors for treatment of rosacea)

L161 ANSWER 15 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:537783 HCAPLUS
DN 129:170541
ED Entered STN: 25 Aug 1998
TI **Nitric oxide** level modulation in treatment of epidermal or dermal conditions
IN Lerner, Ethan A.; Qureshi, Abrar A.; Lerner, Lisa H.
PA The General Hospital Corporation, USA
SO PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A01N037-12
ICS A01N037-44
CC 1-12 (Pharmacology)
Section cross-reference(s): 14, 15
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI WO 9833379 A1 19980806 WO 1998-US1891 19980203
W: AU, CA, JP
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
AU 9861397 A1 19980825 AU 1998-61397 19980203
EP 1001677 A1 20000524 EP 1998-906072 19980203
R: DE, FR, GB, IT
US 6160021 A 20001212 US 1998-18080 19980203
JP 2001511151 T2 20010807 JP 1998-533148 19980203
PRAI US 1997-37098P P 19970204
WO 1998-US1891 W 19980203

AB A method of treating a subject for an unwanted epidermal or dermal condition comprises administering to the subject a treatment which modulates the level of nitric oxide (NO) in the skin. Co-culture of a Langerhans cell-like cell line (XS cells) with melanocytes followed by the induction of nitric oxide synthetase by LPS resulted in melanocyte cell death. LPS had no effect on melanocytes stimulated with LPS in the absence of XS cells. Melanocyte lysis was also seen when cocultures were performed across Transwells, with no direct cell-cell contact between XS cells and melanocytes. The diffusible factor was shown to be NO.

ST **nitric oxide** skin disorder treatment; Langerhans cell **nitric oxide** melanocyte lysis

IT Interleukin 10
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(LPS and IL-10 and **L-NAME** effect on **nitric oxide** synthetase production by Langerhans cells)

IT Skin
(Langerhans' cell; LPS and IL-10 and **L-NAME** effect on **nitric oxide** synthetase production by Langerhans cells)

IT **Skin, disease**
(aging; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT **Skin, disease**
(depigmentation, post-inflammatory hypopigmentation; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT Skin
(dermis; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT Skin
(epidermis; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT Transgene
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(for increasing NO level in skin in animal for evaluating effect of compound of hair or skin; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT Transplant and Transplantation
(graft-vs.-host reaction; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT **Skin, disease**
(hyperpigmentation, post-inflammatory hyperpigmentation; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT **Skin, disease**
(idiopathic guttate hypomelanosis; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT Skin
(keratinocyte, disorder; **nitric oxide** level

modulation in treatment of epidermal or dermal conditions)
IT **Skin, disease**
(lichen planus; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT Dermatitis
Eczema
Psoriasis
Skin, disease
Sunburn
Vitiligo
(**nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT PCR (polymerase chain reaction)
(**nitric oxide** synthetase in murine Langerhans cells; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT Melanocyte
(**nitric oxide** toxicity for; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT Scavengers
(**nitric oxide**; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT Hair
Skin, disease
(pigmentation; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT UV radiation
(skin exposure to; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT **Skin, disease**
(toxic epidermal necrolysis; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT Animal
(transgenic, for evaluating effect of compound on hair or skin; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT Apoptosis
Cell proliferation
(unwanted keratinocyte; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT 211303-24-1 211303-25-2
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(GAPDH PCR primer; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT 50903-99-6, **L-NAME** 67924-63-4, LPS
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(LPS and IL-10 and **L-NAME** effect on **nitric oxide** synthetase production by Langerhans cells)
IT 14402-89-2, Sodium nitroprusside
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(as NO donor; melanocyte lysis by)
IT 125978-95-2, **Nitric oxide** synthase
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); USES (Uses)
(inhibitor; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)

IT 10102-43-9P, **Nitric oxide**, biological studies
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); USES (Uses)
(**nitric oxide** level modulation in treatment of epidermal or dermal conditions)
IT 211303-20-7 211303-21-8 211303-22-9 211303-23-0
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(**nitric oxide** synthetase PCR primer; **nitric oxide** level modulation in treatment of epidermal or dermal conditions)
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
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(4) Zheng; WO 9503810 A1 1995 HCAPLUS

L161 ANSWER 16 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:479406 HCAPLUS
DN 129:86054
ED Entered STN: 03 Aug 1998
TI Pharmaceutical composition for treating fecal incontinence and anal **itch**
IN Kamm, Michael Albert; Phillips, Robin Kenneth Stewart
PA UK
SO PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-00
ICS A61K031-135; A61K031-485; A61K031-195; A61K031-557; A61K031-40
CC 63-6 (Pharmaceuticals)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9827971	A1	19980702	WO 1997-GB3525	19971223
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9853315	A1	19980717	AU 1998-53315	19971223
	AU 728889	B2	20010118		
	EP 946155	A1	19991006	EP 1997-950311	19971223
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, NL, SE, PT, IE, FI				
	JP 2001507020	T2	20010529	JP 1998-528550	19971223
	US 6635678	B1	20031021	US 1999-331163	19990824
	US 2003216420	A1	20031120	US 2003-389773	20030318
PRAI	GB 1996-26739	A	19961223		
	GB 1996-26750	A	19961223		
	GB 1997-3309	A	19970218		
	WO 1997-GB3525	W	19971223		

US 1999-331163 A1 19990824

AB Fecal incontinence and anal itch can be treated by administration, more particularly by local application to the anus, of an α adrenergic blocker, nitric oxide synthase inhibitor, prostaglandin F2α, dopamine, morphine, β-blockers, and 5-Hydroxytryptamine. The patients who benefit most from the invention are those who have a normal or low maximum anal resting pressure and a structurally intact internal anal sphincter muscle, and patients who have had major bowel resection and reanastomosis. Phenylephrine-HCl was added to a base cream to form a composition

ST pharmaceutical fecal incontinence anus **itch**

IT Intestine
(anus; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT Drug delivery systems
(foams; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT Drug delivery systems
(ointments; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT Feces
(pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT Drug delivery systems
(sprays; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT Drug delivery systems
(suppositories; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT Drug delivery systems
(suspensions; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT Adrenoceptor agonists
(α1-; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT Adrenoceptor antagonists
(β-; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; pharmaceutical composition for treating fecal incontinence and anal **itch**)

IT 50-67-9, 5-Hydroxytryptamine, biological studies 51-41-2, Noradrenaline 51-61-6, Dopamine, biological studies 57-27-2, Morphine, biological studies 59-42-7, Phenylephrine 61-76-7, Phenylephrine hydrochloride 390-28-3, Methoxamine 551-11-1, Prostaglandin F2α **2149-70-4**, L-Ornithine, N5-[imino(nitroamino)methyl]- 35700-23-3, Carboprost 50903-99-6, **L-NAME**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pharmaceutical composition for treating fecal incontinence and anal **itch**)

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Bar-Shalom, D; US 5213808 A 1993 HCAPLUS
(2) Burleigh, D; GASTROENTEROLOGY 1983, V84(2), P409 MEDLINE
(3) Byrne, G; US 4292300 A 1981 HCAPLUS
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(5) Enck, P; INTERNIST 1993, V34(1), P51 MEDLINE
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(7) Jauw, T; US 5436009 A 1995

(8) Satish, R; AM J PHYSIOL 1992, V262(1pt1), P107
(9) Shigeru, Y; J CLIN INVEST 1990, V86(2), P424

L161 ANSWER 17 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:456086 HCAPLUS
DN 127:145194
ED Entered STN: 21 Jul 1997
TI Combined use of angiotensin inhibitors and **nitric oxide** stimulators to treat fibrosis
IN Chobanian, Aram; Brecher, Peter
PA Trustees of Boston University, USA
SO U.S., 5 pp.
CODEN: USXXAM
DT Patent
LA English
IC ICM A61K009-00
NCL 424400000
CC 1-12 (Pharmacology)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5645839	A	19970708	US 1995-482819	19950607
	US 6139847	A	20001031	US 1997-801512	19970218
PRAI	US 1995-482819	A3	19950607		

AB A combination of angiotensin inhibitors and nitric oxide stimulators is used to slow and reverse the process of fibrosis in the body. This combination of medicaments is particularly useful in the treatment of a variety of cardiovascular fibrotic pathologies, such as that associated with left ventricular hypertrophy secondary to hypertension, myocardial infarction, and myocarditis.

ST angiotensin **nitric oxide** inhibitor combination fibrosis

IT Respiratory distress syndrome
(adult, fibrosis associated with; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT Angiotensin receptor antagonists
(angiotensin II; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT Antianginal agents
Antiarrhythmics
Anticoagulants
Antihypertensives
Antihypotensives
Diuretics
Fibrosis
Hypolipemic agents
Keloid
Thrombolytics
Vasodilators
(angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT Ion channel blockers
(calcium; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT Glycosides
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cardiac; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT Cardiovascular agents
(cardioplegic; angiotensin inhibitor-**nitric oxide**

stimulator combination for fibrosis treatment)
IT Cardiovascular system
(disease, fibrosis; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Arteriosclerosis
Cirrhosis
Inflammation
(fibrosis associated with; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Lung, disease
(fibrosis; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT **Skin, disease**
(hypertrophic scar; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Heart, disease
(infarction, fibrosis associated with; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Heart, disease
(left ventricle, hypertrophy, secondary to hypertension, fibrosis associated with; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Hypertension
(left ventricular hypertrophy secondary to, fibrosis associated with; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Fibronectins
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(mRNA; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Heart, disease
(myocarditis, fibrosis associated with; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Resins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(potassium-removing; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Ion channel openers
(potassium; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Connective tissue
(scleroderma, fibrosis associated with; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Collagens, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(type III, mRNA; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)
IT Adrenoceptor antagonists
(α-; angiotensin inhibitor- **nitric oxide** stimulator combination for fibrosis treatment)
IT Adrenoceptor antagonists
(β-; angiotensin inhibitor- **nitric oxide** stimulator combination for fibrosis treatment)
IT 74-79-3, L-Arginine, biological studies 50903-99-6, **L-NAME**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 114798-26-4, Losartan
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 55-63-0, Nitroglycerin 78-11-5, Pentaerythritol tetranitrate 87-33-2, Isosorbide dinitrate 139-33-3, Disodium edetate 1002-16-0, Amyl nitrate 15078-28-1, Nitroprusside 62571-86-2, Captopril 74258-86-9, Alacepril 75847-73-3, Enalapril 76420-72-9, Enalaprilat 76547-98-3, Lisinopril 80830-42-8, Rentiapril 81872-10-8, Zofenopril 82834-16-0, Perindopril 82924-03-6, Pentopril 83435-66-9, Delapril 83647-97-6, Spirapril 85441-61-8, Quinapril 87333-19-5, Ramipril 88768-40-5, Cilazapril 98048-97-6, Fosinopril 111223-26-8, Ceranapril
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 11128-99-7, Angiotensin II
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(antagonists and catabolism activators; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 7440-09-7, Potassium, biological studies
RL: BSU (Biological study, unclassified); REM (Removal or disposal); BIOL (Biological study); PROC (Process)
(channel, activators, and potassium-removing resins; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 7440-70-2, Calcium, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(channel, blockers; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 1407-47-2, Angiotensin
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibitors; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 9015-82-1, Angiotensin-converting enzyme 9015-82-1 9025-82-5, Phosphodiesterase 9041-90-1, Angiotensin I
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 85637-73-6, Atrial natriuretic factor
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(mRNA; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 10102-43-9, **Nitric oxide**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(stimulators; angiotensin inhibitor-**nitric oxide** stimulator combination for fibrosis treatment)

IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(stimulators; angiotensin inhibitor-**nitric oxide**

stimulator combination for fibrosis treatment)

L161 ANSWER 18 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:420250 HCAPLUS
DN 127:130624
ED Entered STN: 05 Jul 1997
TI Persistence of effects of **nitric oxide** synthase inhibitors: Comparisons on blood flow and plasma exudation in guinea pig skin
AU Perez, Andrea C.; Khawaja, Aamir M.; Page, Clive P.; Paul, William
CS Department of Pharmacology, King's College London, Manresa Road, London, SW3 6LX, UK
SO European Journal of Pharmacology (1997), 330(2/3), 241-246
CODEN: EJPHAZ; ISSN: 0014-2999
PB Elsevier
DT Journal
LA English
CC 1-7 (Pharmacology)
Section cross-reference(s): 2
AB Plasma protein extravasation has been measured in guinea pig skin using 125I-albumin and blood flow using 133Xenon clearance. The nitric oxide (NO) synthase inhibitors NG-nitro-L-arginine Me ester (L-NAME), NG-monomethyl-L-arginine (L-NMMA) and NG-nitro-L-arginine (L-NOArg) and the α-adrenoceptor agonist, phenylephrine, inhibited bradykinin-induced plasma protein extravasation when co-injected with the peptide. The inhibitory effects of L-NAME and L-NOArg lasted for up to 8 and 4 h, resp., whereas phenylephrine and L-NMMA had no persistent inhibitory effects. When co-injected with 133Xenon, L-NAME, L-NMMA, L-NOArg and phenylephrine, but not D-NAME, produced significant redns. in skin blood flow. When injected prior to 133Xenon, L-NAME and L-NOArg, but not phenylephrine or L-NMMA, significantly reduced flow. The effect of L-NAME on flow was not significant at 8 h. Thus, although the inhibitory effects of the NO synthase inhibitors on mediator-induced plasma protein extravasation show correlations with their effects on blood flow, the persistent effect of L-NAME on exudation appears to extend beyond its effect on flow.
ST **nitric oxide** synthase inhibitor skin circulation; skin plasma exudation edema NOS inhibitor
IT **Skin, disease**
Skin, disease
(edema; **nitric oxide** synthase inhibitors effects on blood flow and plasma exudation in skin)
IT Blood plasma
(exudation; **nitric oxide** synthase inhibitors effects on blood flow and plasma exudation in skin)
IT Circulation
(skin; **nitric oxide** synthase inhibitors effects on blood flow and plasma exudation in skin)
IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; **nitric oxide** synthase inhibitors effects on blood flow and plasma exudation in skin)
IT 141968-19-6, D-**NAME**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**nitric oxide** synthase inhibitors effects on blood flow and plasma exudation in skin)
IT 59-42-7, Phenylephrine 2149-70-4, **NG-Nitro-L-arginine** 17035-90-4, **L-NMMA** 50903-99-6, **L-NAME**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**nitric oxide** synthase inhibitors effects on blood flow and plasma exudation in skin)

IT 10102-43-9, **Nitric oxide**, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**nitric oxide** synthase inhibitors effects on blood flow and plasma exudation in skin)

RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L161 ANSWER 19 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:92315 HCAPLUS
DN 126:126704
ED Entered STN: 08 Feb 1997
TI L-Arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice
AU Aji, Walif; Ravalli, Stefano; Szabolcs, Matthias; Jiang, Xian-Cheng; Sciacca, Robert R.; Michler, Robert E.; Cannon, Paul J.
CS Department of Medicine, Columbia University College of Physicians and Surgeons, New York, NY, USA
SO Circulation (1997), 95(2), 430-437
CODEN: CIRCAZ; ISSN: 0009-7322
PB American Heart Association
DT Journal
LA English
CC 1-8 (Pharmacology)
AB The potential antiatherosclerotic actions of NO were investigated in four groups of mice (n = 10 per group) lacking functional LDL receptor genes, an animal model of familial hypercholesterolemia. Group I was fed a regular chow diet. Groups 2 through 4 were fed a 1.25% high-cholesterol diet. In addition, group 3 received supplemental L-arginine and group 4 received L-arginine and Nω-nitro-L-arginine (L-NA), an inhibitor of NO synthase (NOS). Animals were killed at 6 mo; aortas were stained with oil red O for planimetry and with antibodies against constitutive and inducible NOSs. Plasma cholesterol was markedly increased in the animals receiving the high-cholesterol diet. Xanthomas appeared in all mice fed the high-cholesterol diet alone but not in those receiving L-arginine.

Aortic atherosclerosis was present in all mice on the high-cholesterol diet. The mean atherosclerotic lesion area was reduced significantly (P<.01) in the cholesterol-fed mice given L-arginine compared with those receiving the high-cholesterol diet alone. The mean atherosclerotic lesion area was significantly larger (P<.01) in cholesterol-fed mice receiving L-arginine + L-NA than in those on the high-cholesterol diet alone. Within the atherosclerotic plaques, endothelial cells immunoreacted for endothelial cell NOS; macrophages, foam cells, and smooth muscle cells immunostained strongly for inducible NOS and nitrotyrosine residues. The data indicate that L-arginine prevents xanthoma formation and reduces atherosclerosis in LDL receptor knockout mice fed a high-cholesterol diet. The abrogation of the beneficial effects of L-arginine by L-NA suggests that the anti-atherosclerotic actions of L-arginine are mediated by NOS. The data suggest that L-arginine may be beneficial in familial hypercholesterolemia.

ST arginine xanthoma atherosclerosis LDL receptor knockout; antiatherosclerotic arginine **nitric oxide**

IT Lipoprotein receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(LDL; arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice)

IT Antiarteriosclerotics
(antiatherosclerotics; arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice)

IT Hypercholesterolemia
(familial; arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice)

IT **Skin, disease**
(xanthoma; arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice)

IT 2149-70-4, **Nω -Nitro-L-arginine**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice)

IT 10102-43-9, **Nitric oxide**, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice)

IT 74-79-3, L-Arginine, biological studies
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice)

IT 125978-95-2, **Nitric oxide** synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitor; arginine prevents xanthoma development and inhibits atherosclerosis in LDL receptor knockout mice)

RE.CNT 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L161 ANSWER 20 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1996:597098 HCAPLUS
DN 125:238483
ED Entered STN: 07 Oct 1996
TI Treatment of a chronic allodynia-like response in spinally injured rats: Effects of systemically administered **nitric oxide** synthase inhibitors
AU Hao, Jing-Xia; Xu, Xiao-Jun
CS Karolinska Institute, Huddinge University Hospital, Huddinge, Swed.
SO Pain (1996), 66(2,3), 313-319

CODEN: PAINDB; ISSN: 0304-3959
PB Elsevier
DT Journal
LA English
CC 1-11 (Pharmacology)
AB We have previously reported that we have observed chronic pain-like response to light mech. stimuli (allodynia) in rats after severe spinal cord ischemia, which resembles some painful conditions in chronic spinally injured patients and is not relieved by a number of conventional analgesics used for treating chronic neuropathic pain. In the present study, we tested the effects of the non-selective nitric oxide synthase (NOS) inhibitor NG-nitro-L-arginine Me ester (L-NAME) and the selective neuronal NOS inhibitor 7-nitro indazole (7-NI) and 6-nitro indazole (6-NI) on the chronic allodynia-like behavior. Systemic L-NAME dose-dependently relieved mech. allodynia-like response in a stereo-specific and L-arginine-reversible manner without causing sedation or motor deficits. However, L-NAME significantly elevated systemic blood pressure. Systemic 7-NI relieved chronic allodynia in a L-arginine reversible manner, did not increase blood pressure or induce sedation, but caused motor deficits at a high dose, which was not reversed by L-arginine. Systemic 6-NI also relieved the chronic allodynia, which was however associated with severe sedation. To exclude the possibility that the effect of L-NAME on blood pressure was involved in the analgesic effect observed, the effect of systemically applied adrenaline was examined Adrenaline increased the systemic blood pressure to a similar extent as L-NAME, but did not relieve allodynia. It is suggested that blockade of NOS by L-NAME relieved the chronic allodynia-like behavior in spinally injured rats. This effect was likely to be mediated by a blockade of neuronal isoforms of NOS, as 7-NI relieved the allodynia in a L-arginine-reversible manner. Consequently, generation of NO by neuronal NOS may be critically involved in the maintenance of this abnormal pain-related sensation. The possibility of using NOS inhibitors as potential novel analgesics is discussed.
ST allodynia spinal injury NOS inhibitor analgesic; **nitric oxide** synthase allodynia spinal injury
IT Analgesics
(treatment of a chronic allodynia-like response in spinally injured rats: effects of systemically administered **nitric oxide** synthase inhibitors)
IT **Skin, disease**
(allodynia, treatment of a chronic allodynia-like response in spinally injured rats: effects of systemically administered **nitric oxide** synthase inhibitors)
IT Spinal cord
(disease, injury, treatment of a chronic allodynia-like response in spinally injured rats: effects of systemically administered **nitric oxide** synthase inhibitors)
IT 125978-95-2, **Nitric oxide** synthase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibitors; treatment of a chronic allodynia-like response in spinally injured rats: effects of systemically administered **nitric oxide** synthase inhibitors)
IT 2942-42-9, 7-Nitro **indazole** 7597-18-4, 6-Nitro **indazole** 50903-99-6, **L-NAME**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(treatment of a chronic allodynia-like response in spinally injured rats: effects of systemically administered **nitric oxide** synthase inhibitors)

IT 10102-43-9, **Nitric oxide**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(treatment of a chronic allodynia-like response in spinally injured rats: effects of systemically administered **nitric oxide** synthase inhibitors)

L161 ANSWER 21 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1996:393980 HCAPLUS
DN 125:49348
ED Entered STN: 10 Jul 1996
TI Regulation of wound healing by **nitric oxide**
IN Murrell, George Anthony Calvert
PA New York Society for the Ruptured and Crippled Maintaining the Hospital for Special Surgery, USA
SO PCT Int. Appl., 42 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A01N033-00
CC 1-12 (Pharmacology)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9608966	A1	19960328	WO 1995-US12503	19950921
	W: CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 6190704	B1	20010220	US 1994-311545	19940923
PRAI	US 1994-311545	A	19940923		

AB A method for enhancement of wound healing by exposure to increased levels of nitric oxide is disclosed. Also disclosed is a method for inhibition of wound healing by decreasing the level of nitric oxide in a wound.
ST **nitric oxide** wound healing regulation
IT Fibroblast
(identification of agents inhibiting unwanted fibroblast proliferation in damaged tissue)
IT Wound healing
(**nitric oxide** for regulation of wound healing)
IT Tendon
(Achilles, identification of agents inhibiting unwanted fibroblast proliferation in damaged tissue)
IT Bone, disease
(injury, **nitric oxide** for regulation of wound healing)
IT Muscle, disease
Skin, disease
(wound, **nitric oxide** for regulation of wound healing)
IT 50903-99-6, **L-NAME**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**nitric oxide** for regulation of wound healing)
IT 125978-95-2, **Nitric oxide** synthase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**nitric oxide** for regulation of wound healing)
IT 10102-43-9, **Nitric oxide**, biological studies
17035-90-4
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(**nitric oxide** for regulation of wound healing)

L161 ANSWER 22 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1996:58971 HCAPLUS
DN 124:172436
ED Entered STN: 30 Jan 1996
TI **L-NAME**, an inhibitor of **nitric oxide** synthase, blocks the established allodynia induced by intrathecal administration of prostaglandin E2
AU Minami, Toshiaki; Onaka, Masahiko; Okuda-Ashitaka, Emiko; Mori, Hidemaro; Ito, Seiji; Hayaishi, Osamu
CS Department of Anesthesiology, Osaka Medical College, Takatsuki, 569, Japan
SO Neuroscience Letters (1995), 201(3), 239-42
CODEN: NELED5; ISSN: 0304-3940
PB Elsevier
DT Journal
LA English
CC 14-9 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 2
AB We recently reported that intrathecal (i.t.) administration of prostaglandin E2 (PGE2) to conscious mice induced allodynia, a state of discomfort and pain evoked by innocuous tactile stimuli. In the present study, we examined the effect of the PGE receptor EP1 subtype antagonist ONO-NT-012, the N-methyl-D-aspartate (NMDA) receptor antagonist MK-801, and the NO synthase inhibitor Nω-nitro-L-arginine Me ester (L-NAME) on the allodynia. The PGE2-induced allodynia was blocked by simultaneous i.t. injection of ONO-NT-012, MK-801, or L-NAME. However, 5 min after i.t. injection of PGE2, the allodynia was significantly blocked by i.t. L-NAME, but not by i.t. ONO-NT-012 or MK-801. These results demonstrate that the PGE2-induced allodynia, once developed, does not require the continued agonist occupancy of EP1 and NMDA glutamate receptor sites.
ST prostaglandin allodynia EP1 glutamate receptor
IT Prostaglandin receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PGE2-induced allodynia, once developed, does not require the continued agonist occupancy of EP1 and NMDA glutamate receptor sites)
IT **Skin, disease**
(allodynia, PGE2-induced allodynia, once developed, does not require the continued agonist occupancy of EP1 and NMDA glutamate receptor sites)
IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(glutamatergic, PGE2-induced allodynia, once developed, does not require the continued agonist occupancy of EP1 and NMDA glutamate receptor sites)
IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(prostaglandin, PGE2-induced allodynia, once developed, does not require the continued agonist occupancy of EP1 and NMDA glutamate receptor sites)
IT 363-24-6, Prostaglandin E2 50903-99-6, **L-NAME**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(PGE2-induced allodynia, once developed, does not require the continued agonist occupancy of EP1 and NMDA glutamate receptor sites)

L161 ANSWER 23 OF 23 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1994:70119 HCAPLUS
DN 120:70119
ED Entered STN: 19 Feb 1994

TI Role of prostaglandins and **nitric oxide** in acute inflammatory reactions in guinea pig skin
AU Teixeira, M. M.; Williams, T. J.; Hellewell, P. G.
CS Dep. Appl. Pharmacol., Natl. Heart Lung Inst., London, SW3 6LY, UK
SO British Journal of Pharmacology (1993), 110(4), 1515-21
CODEN: BJPCBM; ISSN: 0007-1188
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
Section cross-reference(s): 15
AB Edema formation in skin is dependent on a synergism between mediators that increase vascular permeability and mediators that enhance local blood flow. Leukocyte accumulation is also enhanced by mediators that increase local blood flow. In this study, the authors have investigated whether nitric oxide (NO), an important endogenous vasodilator, could modulate edema formation and leukocyte accumulation in guinea-pig skin. Local administration of the NO synthesis inhibitor NG-nitro-L-arginine Me ester (L-NAME) dose-dependently inhibited the edema formation induced in response to intradermal injection of bradykinin or histamine. L-NAME, but not NG-nitro-D-arginine Me ester (D-NAME), also inhibited edema formation in response to i.d. injection of platelet-activating factor (PAF), zymosan-activated plasma (ZAP) and in a passive cutaneous anaphylactic (PCA) reaction. N-iminoethyl-L-ornithine (L-NIO) was less effective and about 100 times less potent than L-NAME in inhibiting bradykinin-induced edema formation. The cyclooxygenase inhibitor ibuprofen had little effect on edema responses induced by bradykinin, PAF and in a PCA reaction. Histamine-induced edema formation was significantly suppressed by ibuprofen. The inhibition by L-NAME of bradykinin-induced edema formation was reversed by co-injection of sodium nitroprusside (SNP) or prostaglandin E1 (PGE1). L-NAME inhibited 111In-eosinophil and 111In-neutrophil accumulation induced by i.d. injection of ZAP. 111In-eosinophil accumulation induced by PAF and in the PCA reaction was also inhibited by L-NAME but not by D-NAME. Co-injection of SNP or PGE1 reversed the inhibition by L-NAME of ZAP-induced edema formation and 111In-neutrophil accumulation. SNP, but not PGE1, also reversed the effects of L-NAME on ZAP-induced 111In-eosinophil accumulation. L-NAME caused a significant decrease in basal cutaneous blood flow when injected alone or with bradykinin. Again, SNP or PGE1 reversed the effects of L-NAME suggesting that the inhibitory action of L-NAME on edema formation and cell accumulation was due to an inhibition of vasodilator tone in the microcirculation. Thus, it appears that in guinea-pig skin the inhibition of the production of endogenous NO inhibits both leukocyte accumulation and edema formation induced by different mediators of inflammation. Since administration of L-NAME also causes a local decrease in basal blood flow, the authors suggest that this is the mechanism by which it exerts anti-inflammatory effects in this model.
ST skin inflammation prostaglandin **nitric oxide**; leukocyte accumulation inflammation prostaglandin **nitric oxide**; edema inflammation prostaglandin **nitric oxide**
IT Eosinophil
Neutrophil
(accumulation of, in acute inflammatory reactions in guinea pig skin, **L-NAME** inhibition of)
IT Leukocyte
(accumulation of, **nitric oxide** and prostaglandins modulation of, in acute inflammatory reactions in guinea pig skin)
IT Inflammation inhibitors
(acute inflammatory reactions in guinea pig skin inhibition by, **nitric oxide** and prostaglandins in relation to)

IT Prostaglandins
RL: BIOL (Biological study)
(acute inflammatory reactions in guinea pig skin modulation by **nitric oxide** and)
IT Guinea pig
(acute inflammatory reactions in skin of, **nitric oxide** and prostaglandins modulation of)
IT Circulation
(in skin, **L-NAME** effect on, **nitric oxide** and prostaglandins modulation of acute inflammatory reactions in relation to)
IT Dermatitis
(**nitric oxide** and prostaglandins modulation of, in guinea pig)
IT **Skin, disease**
(edema, formation of, in acute inflammatory reactions in guinea pig, **nitric oxide** and prostaglandins modulation of)
IT 50903-99-6, **L-NAME**
RL: BIOL (Biological study)
(acute inflammatory reactions in guinea pig skin inhibition by, **nitric oxide** and prostaglandins in relation to)
IT 10102-43-9, **Nitric oxide**, biological studies
RL: BIOL (Biological study)
(acute inflammatory reactions in guinea pig skin modulation by prostaglandins and)
IT 36889-13-1
RL: BIOL (Biological study)
(bradykinin-induced edema formation in guinea pig skin inhibition by)
IT 51-45-6, Histamine, biological studies 58-82-2, Bradykinin 65154-06-5, Platelet-activating factor
RL: BIOL (Biological study)
(skin edema in guinea pig induced by, **L-NAME** effect on, **nitric oxide** and prostaglandins modulation of acute inflammatory reactions in relation to)

=> b medline

FILE 'MEDLINE' ENTERED AT 10:45:40 ON 03 MAR 2004

FILE LAST UPDATED: 2 MAR 2004 (20040302/UP). FILE COVERS 1953 TO DATE.

On February 29, 2004, the 2004 MeSH terms were loaded. See HELP RLOAD for details.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2004 vocabulary. See http://www.nlm.nih.gov/mesh/ and http://www.nlm.nih.gov/pubs/techbull/nd03/nd03_mesh.html for a description of changes.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d all l107 8 14 15 17 25 26 37

L107 ANSWER 8 OF 47 MEDLINE on STN
AN 2003015534 MEDLINE
DN PubMed ID: 12522091
TI **Nitric oxide** enhances substance P-induced **itch**

-associated responses in mice.

AU Andoh Tsugunobu; Kuraishi Yasushi
CS Department of Applied Pharmacology, Faculty of Pharmaceutical Sciences, Toyama Medical and Pharmaceutical University, 2630 Sugitani, Toyama 930-0194, Japan.

SO British journal of pharmacology, (2003 Jan) 138 (1) 202-8.
Journal code: 7502536. ISSN: 0007-1188.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200307
ED Entered STN: 20030111
Last Updated on STN: 20030716
Entered Medline: 20030715

AB 1 Substance P (SP) elicits **itch** and **itch**-associated responses in humans and mice, respectively. In mice, NK(1) tachykinin receptors are involved in SP-induced **itch**-associated responses, **scratching**, and mast cells do not play a critical role. The present study was conducted to elucidate the role of **nitric oxide** (NO) on SP-induced **scratching** in mice. 2 An intradermal injection of SP (100 nmol site(-1)) elicited **scratching** in mice, and it was suppressed by an intravenous injection of the NO synthase (NOS) inhibitor N(G)-**nitro**-**L-arginine** methyl ester (**L-NAME**), but not by its inactive enantiomer D-**NAME**. Intradermal injections of **L-NAME** (100 nmol site(-1)), another NOS inhibitor **7-nitroindazole** (10 nmol site(-1)) and the NO scavenger haemoglobin (0.01-10 nmol site(-1)) also inhibited SP-induced **scratching**. 3 **L-NAME** (100 nmol site(-1)) did not affect **scratching** induced by an intradermal injection of 5-hydroxytryptamine (100 nmol site(-1)). 4 Intradermal injections of L-arginine (300 nmol site(-1)) and the NO donor (+/-)-(E)-4-ethyl-2-[(E)-hydroxyimino]-5-nitro-3-hexenamide (NOR3; 100 nmol site(-1)) increased **scratching** induced by SP. Intradermal injections of L-arginine (1-1000 nmol site(-1)) or NOR3 (1-100 nmol site(-1)) alone were without effects on **scratching**. 5 Intradermal injections of SP (10-100 nmol site(-1)) increased the intradermal concentration of NO in a dose-dependent manner in mice. An increase in NO levels induced by SP was inhibited by **L-NAME** and the NK(1) tachykinin receptor antagonist L-668,169, but not by the NK(2) tachykinin receptor antagonist L-659,877. 6 SP (1-10 micro M) elicited NO production in cultured human keratinocytes and the SP-induced NO production was inhibited by **L-NAME** and L-668,169. 7 We conclude that intradermal SP increases NO in the skin, possibly through the action on NK(1) tachykinin receptors on the epidermal keratinocytes and that NO enhances SP-induced **itch**-associated responses.

CT Check Tags: Human; Male; Support, Non-U.S. Gov't
Adult
Animals
Enzyme Inhibitors: PD, pharmacology
Enzyme Inhibitors: TU, therapeutic use
Mice
Mice, Inbred ICR
***Nitric Oxide: BI, biosynthesis**
Nitric-Oxide Synthase: AI, antagonists & inhibitors
Nitric-Oxide Synthase: ME, metabolism
***Pruritus: CI, chemically induced**
Pruritus: DT, drug therapy
Pruritus: EN, enzymology

Pruritus: ME, metabolism
Receptors, Neurokinin-1: AI, antagonists & inhibitors
Receptors, Neurokinin-1: ME, metabolism
*Substance P: TO, toxicity
RN **10102-43-9 (Nitric Oxide)**; 33507-63-0 (Substance P)
CN 0 (Enzyme Inhibitors); 0 (Receptors, Neurokinin-1); EC 1.14.13.39 (**Nitric-Oxide** Synthase)

L107 ANSWER 14 OF 47 MEDLINE on STN
AN 2000271662 MEDLINE
DN PubMed ID: 10813554
TI The role of **nitric oxide** and prostaglandin E2 on the hyperalgesia induced by excitatory amino acids in rats.
AU Park Y H; Shin C Y; Lee T S; Huh I H; Sohn U D
CS Department of Pharmacology, School of Pharmacy, Chung Ang University, Seoul, Republic of Korea.
SO Journal of pharmacy and pharmacology, (2000 Apr) 52 (4) 431-6.
Journal code: 0376363. ISSN: 0022-3573.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200007
ED Entered STN: 20000720
Last Updated on STN: 20000720
Entered Medline: 20000712
AB The present study was designed to investigate the role of **nitric oxide** (NO), N-methyl-D-aspartate (NMDA) receptor and prostaglandins on hyperalgesia induced in rats by excitatory amino acids and the possibility that prostaglandins may act as the retrograde messenger in the spinal cord like NO. Nomega-**nitro**-**L-arginine** methyl ester (**L-NAME**; 500 microg/paw, intraplantarly (i.pl.)), MK-801 (10 microg/paw, i.pl.) or indomethacin (300 microg/paw, i.pl.) reduced the duration of phase 2 of the biting/licking and **scratching** (B/L + S) response induced by formalin injection from 255.6+/-16.7 s to 155.6+/-16.9, 172.25+/-33.3 or 205.6+/-16.7 s, respectively. **L-NAME** (0.3 mg, i.th.), MK-801 (8 microg, i.th.) or indomethacin (20 microg, i.th) reduced the duration of phase 2 of the B/L + S response induced by saline injection from 288.5+/-7.7s to 207.7+/-19.2, 184.6+/-7.7 or 1923+/-38.5 s, respectively. **L-NAME** or indomethacin injected into the spinal cord of the rat significantly reduced the hyperalgesia induced by NMDA (1 microg, i.th.) from 43.8+/-4.6% to 12.3+/-3.1 and 19.2+/-2.3%, respectively. It is assumed that NO produced by excitatory amino acids may increase prostaglandin production by cyclooxygenase activation. **L-NAME**, MK-801 or indomethacin injected into the rat spinal cord significantly reduced the hyperalgesia induced by prostaglandin E2 (PGE2, 25 ng, i.th.) in the tail-flick test from 40.6+/-3.5% to 18.2+/-3.2, 18.8+/-1.8 or 17.6+/-4.1%, respectively, but had little effect on hyperalgesia in the paw pressure test (except for indomethacin). In conclusion, NO and PGE2 affect the hyperalgesia induced by excitatory amino acids. It is suggested that PGE2, like NO, may act as a retrograde messenger in the spinal cord.
CT Check Tags: Male; Support, Non-U.S. Gov't
Animals
Dinoprostone: AD, administration & dosage
*Dinoprostone: PH, physiology
Dizocilpine Maleate: PD, pharmacology
*Excitatory Amino Acids: AD, administration & dosage
Formaldehyde: AD, administration & dosage

Hyperalgesia: CI, chemically induced
*Hyperalgesia: PP, physiopathology
Hyperalgesia: PC, prevention & control
Indomethacin: PD, pharmacology
N-Methylaspartate: AD, administration & dosage
NG-Nitroarginine Methyl Ester: PD, pharmacology
***Nitric Oxide: PH, physiology**
Pain: CI, chemically induced
Pain: PP, physiopathology
Pain: PC, prevention & control
Pain Measurement
Rats
Rats, Sprague-Dawley
Time Factors

RN **10102-43-9 (Nitric Oxide)**; 363-24-6 (Dinoprostone); 50-00-0 (Formaldehyde); **50903-99-6 (NG-Nitroarginine Methyl Ester)**; 53-86-1 (Indomethacin); 6384-92-5 (N-Methylaspartate); 77086-22-7 (Dizocilpine Maleate)

CN 0 (Excitatory Amino Acids)

L107 ANSWER 15 OF 47 MEDLINE on STN

AN 2000095313 MEDLINE

DN PubMed ID: 10629849

TI Involvement of **nitric oxide** in **itch-scratch** response of NC mice.

AU Tsukumo Y; Andoh T; Yamaguchi T; Nojima H; Kuraishi Y

CS Department of Applied Pharmacology, Faculty of Pharmaceutical Sciences, Toyama Medical and Pharmaceutical University, Japan.

SO Nippon yakurigaku zasshi. Japanese journal of pharmacology, (1999 Oct) 114 Suppl 1 17P-21P.
Journal code: 0420550. ISSN: 0015-5691.

CY Japan

DT Journal; Article; (JOURNAL ARTICLE)

LA Japanese

FS Priority Journals

EM 200002

ED Entered STN: 20000229
Last Updated on STN: 20000229
Entered Medline: 20000216

AB NC mice, a model for atopic dermatitis, showed **scratching** behavior when kept under conventional environment. The **scratching** behavior of NC mice was suppressed by distraction or by the administration of naltrexone (1 mg/kg, s.c.), an opioid antagonist. These results suggest that such **scratching** behavior is **itch**-associated response. The **itch**-associated response of the NC mice was significantly suppressed by an intravenous injection of **nitric oxide** (NO) synthase inhibitor **NG-nitro-L-arginine** methyl ester (**L-NAME**, 10 mg/kg), but not D-**NAME** (10 mg/kg) and saline. Intracutaneous NO production in the rostral back, a region which the NC mice mainly **scratched**, was markedly increased as compared with the caudal back, a non-**scratched** region. The increased NO production in the rostral back of NC mice was decreased by the intravenous injection of **L-NAME** (10 mg/kg). These results suggest that NO and NO synthase are new target in the treatment of atopic **pruritus.**

CT Check Tags: Female; Male
Animals
*Dermatitis, Atopic: PP, physiopathology
Disease Models, Animal

English Abstract
Enzyme Inhibitors: PD, pharmacology
Mice
Mice, Inbred Strains
NG-Nitroarginine Methyl Ester: PD, pharmacology
***Nitric Oxide: PH, physiology**
Nitric-Oxide Synthase: AI, antagonists & inhibitors
***Pruritus: PP, physiopathology**

RN **10102-43-9 (Nitric Oxide); 50903-99-6 (NG-Nitroarginine Methyl Ester)**
CN 0 (Enzyme Inhibitors); EC 1.14.13.39 (**Nitric-Oxide** Synthase)

L107 ANSWER 17 OF 47 MEDLINE on STN
AN 1999386754 MEDLINE
DN PubMed ID: 10455330
TI Nociceptin-induced **scratching**, biting and licking in mice: involvement of spinal NK1 receptors.
AU Sakurada T; Katsuyama S; Sakurada S; Inoue M; Tan-No K; Kisara K; Sakurada C; Ueda H; Sasaki J
CS Department of Biochemistry, Daiichi College of Pharmaceutical Sciences, 22-1 Tamagawa-cho, Minami-ku, Fukuoka 815-8511, Japan.
SO British journal of pharmacology, (1999 Aug) 127 (7) 1712-8.
Journal code: 7502536. ISSN: 0007-1188.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
ED Entered STN: 19991026
Last Updated on STN: 19991026
Entered Medline: 19991014
AB 1. Intrathecal (i.t.) injection of nociceptin at small doses (fmol order) elicited a behavioural response consisting of **scratching**, biting and licking in conscious mice. Here we have examined the involvement of substance P-containing neurons by using i.t. injection of tachykinin neurokinin (NK)1 receptor antagonists and substance P (SP) antiserum. 2. Nociceptin-induced behavioural response was evoked significantly 5 - 10 min after i.t. injection and reached a maximum at 10 - 15 min. Dose-dependency of the induced response showed a bell-shaped pattern from 0.375 - 30.0 fmol, and the maximum effect was observed at 3.0 fmol. 3. The behavioural response elicited by nociceptin (3.0 fmol) was dose-dependently inhibited by intraperitoneal (i.p.) administration of morphine. 4. The NK1 receptor antagonists, CP-96,345, CP-99,994 and sendide, inhibited nociceptin-induced behavioural response in a dose-dependent manner. A significant antagonistic effect of [D-Phe7, D-His9]SP (6 - 11), a selective antagonist for SP receptors, was observed against nociceptin-induced response. The NK2 receptor antagonist, MEN-10376, had no effect on the response elicited by nociceptin. 5. Pretreatment with SP antiserum resulted in a significant reduction of the response to nociceptin. No significant reduction of nociceptin-induced response was detected in mice pretreated with NKA antiserum. 6. The N-methyl-D-aspartate (NMDA) receptor antagonists, dizocilpine (MK-801) and D(-)-2-amino-5-phosphonovaleric acid (APV) (D-APV), and L-NG-nitro arginine methyl ester (**L-NAME**), a **nitric oxide** (NO) synthase inhibitor, failed to inhibit nociceptin-induced behavioural response. 7. off present results suggest that SP-containing neurons in the mouse spinal cord may be involved in elicitation of **scratching**, biting and licking behaviour following i.t. injection of nociceptin.

CT Check Tags: Male
*Aggression: DE, drug effects
Analgesics, Opioid: AD, administration & dosage
Analgesics, Opioid: PD, pharmacology
Animals
Antibodies, Blocking: PD, pharmacology
*Behavior, Animal: DE, drug effects
Biphenyl Compounds: PD, pharmacology
Dose-Response Relationship, Drug
Injections, Spinal
Mice
Morphine: AD, administration & dosage
Morphine: PD, pharmacology
Neurokinin A: AI, antagonists & inhibitors
Neurons: DE, drug effects
Neurons: PH, physiology
Opioid Peptides: AD, administration & dosage
Opioid Peptides: AI, antagonists & inhibitors
*Opioid Peptides: PD, pharmacology
Piperidines: PD, pharmacology
Receptors, Neurokinin-1: AI, antagonists & inhibitors
*Receptors, Neurokinin-1: DE, drug effects
*Receptors, Opioid: AG, agonists
Spinal Cord: DE, drug effects
*Spinal Cord: ME, metabolism
Substance P: AI, antagonists & inhibitors
Substance P: IM, immunology
Substance P: PH, physiology
RN 132746-60-2 (CP 96345); 136982-36-0 (3-(2-methoxybenzylamino)-2-phenylpiperidine); 33507-63-0 (Substance P); 57-27-2 (Morphine); 86933-74-6 (Neurokinin A)
CN 0 (Analgesics, Opioid); 0 (Antibodies, Blocking); 0 (Biphenyl Compounds); 0 (Opioid Peptides); 0 (Piperidines); 0 (Receptors, Neurokinin-1); 0 (Receptors, Opioid); 0 (nociceptin)

L107 ANSWER 25 OF 47 MEDLINE on STN
AN 1998312846 MEDLINE
DN PubMed ID: 9650826
TI Basal **nitric oxide** release differentially modulates vasodilations by pinacidil and levcromakalim in goat coronary artery.
AU Deka D K; Raviprakash V; Mishra S K
CS Division of Pharmacology and Toxicology, Indian Veterinary Research Institute, Izatnagar, UP.
SO European journal of pharmacology, (1998 May 1) 348 (1) 11-23.
Journal code: 1254354. ISSN: 0014-2999.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199809
ED Entered STN: 19980925
Last Updated on STN: 19990129
Entered Medline: 19980911
AB In the current investigation, the role of basal **nitric oxide** (NO) in modulating the vasorelaxant responses to pinacidil and levcromakalim was examined in goat isolated coronary artery. Pinacidil (10(-8) 10(-4) M) elicited concentration-dependent relaxations of the coronary artery ring segments (with intact endothelium) constricted with 30 mM K+ saline solution. The EC50 of the vasodilator was 2.57 x 10(-6) M (95% CL, 1.9-3.46 x 10(-6) M). The removal of endothelium by

mechanical **rubbing** caused a rightward shift in the concentration-response curve of pinacidil with a corresponding increase in EC50 value (1.90 x 10(-5) M; 95% CL, 1.12-3.23 x 10(-5) M). Similar to endothelium removal, treatment of endothelium-intact rings either with the NO synthesis inhibitor **L-NAME** (**NG-nitro-L-arginine** methyl ester; 3 x 10(-5) M) or the guanylate cyclase inhibitor, methylene blue (3 x 10(-6) M) resulted in a marked inhibition in the relaxant responses to pinacidil. Hence, the EC50 values of the potassium channel opener were significantly higher in tissues treated either with **L-NAME** (7.41 x 10(-6) M; 95% CL, 6.02-9.12 x 10(-6) M) or methylene blue (2.29 x 10(-5) M; 95% CL, 1.58-3.31 x 109-5) M) as compared to untreated controls. The ATP-sensitive potassium (KATP) channel blocker glibenclamide, which caused a significant rightward shift in the concentration-relaxation curve of pinacidil in control tissues, was found to be less potent in antagonising the relaxant responses of the KATP channel opener in endothelium-denuded rings and in rings with intact endothelium but treated with either **L-NAME** or methylene blue. In contrast to the observations made with pinacidil, the vasodilator responses to another KATP channel opener, levcromakalim, were potentiated in the absence of basal NO. Thus, the EC50 of levcromakalim was 1.33 x 10(-8) M (95% CL, 0.8-2.21 x 10(-8) M) in control tissues with intact endothelium, which was significantly higher than those obtained in endothelium-deprived rings (4.81 x 10(-9) M; 95% CL, 4.04-5.73 x 10(-9) M) or endothelium intact rings treated either with **L-NAME** (2.63 x 10(-9) M; 95% CL, 1.58-4.36 x 10(-9) M) or methylene blue (2.82 x 10(-9) M; 95% CL, 1.7-4.68 x 10(-9) M). The selective modulation by basal NO of the arterial relaxations elicited with the KATP channel openers was evident from the findings that papaverine-induced relaxations were not affected in the absence of basal NO. Taken together, the results of the present study suggest that basal NO differentially modulates the interaction of pinacidil and levcromakalim with the KATP channels in goat coronary artery through a cGMP-dependent pathway.

CT Check Tags: In Vitro
Animals
*Coronary Vessels: DE, drug effects
*Cromakalim: PD, pharmacology
Endothelium, Vascular: DE, drug effects
Enzyme Inhibitors: PD, pharmacology
Glyburide: PD, pharmacology
Goats
*Guanidines: PD, pharmacology
Hypoglycemic Agents: PD, pharmacology
Methylene Blue: PD, pharmacology
NG-Nitroarginine Methyl Ester: PD, pharmacology
***Nitric Oxide: ME, metabolism**
Nitric Oxide: PH, physiology
Nitric-Oxide Synthase: AI, antagonists & inhibitors
Papaverine: PD, pharmacology
Pinacidil
*Potassium Channels: AG, agonists
*Vasodilation: DE, drug effects
Vasodilation: PH, physiology
*Vasodilator Agents: PD, pharmacology

RN **10102-43-9 (Nitric Oxide)**; 10238-21-8 (Glyburide); **50903-99-6 (NG-Nitroarginine Methyl Ester)**; 58-74-2 (Papaverine); 61-73-4 (Methylene Blue); 85371-64-8 (Pinacidil); 94470-67-4 (Cromakalim)

CN 0 (Enzyme Inhibitors); 0 (Guanidines); 0 (Hypoglycemic Agents); 0 (Potassium Channels); 0 (Vasodilator Agents); EC 1.14.13.39 (**Nitric-Oxide** Synthase)

L107 ANSWER 26 OF 47 MEDLINE on STN
AN 1998305768 MEDLINE
DN PubMed ID: 9643615
TI Endothelin-1 induces vasodilation in human skin by nociceptor fibres and release of **nitric oxide.**
AU Wenzel R R; Zbinden S; Noll G; Meier B; Luscher T F
CS Cardiology, Cardiovascular Research, University Hospital, Inselspital, Bern, Switzerland.
SO British journal of clinical pharmacology, (1998 May) 45 (5) 441-6.
Journal code: 7503323. ISSN: 0306-5251.
CY ENGLAND: United Kingdom
DT (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199809
ED Entered STN: 19980910
Last Updated on STN: 19980910
Entered Medline: 19980901
AB AIMS: Endothelin is a peptide produced by endothelial cells with many biological properties. In the human skin microcirculation endothelin induces neurogenic vasodilation associated with burning **pruritus**. We investigated the mechanisms involved in this response. METHODS: The effects of prolonged pretreatment with capsaicin, a specific inhibitor of polimodal nociceptor fibres, and of the **nitric oxide** synthase inhibitor **L-NMMA** on endothelin-1-induced vasodilation were studied in 15 human subjects. Furthermore, we investigated the effects of the ET(A)-selective antagonist PD147953 on bradykinin-induced vasodilation. RESULTS: After local injection, endothelin-1 caused vasoconstriction at the injection site and a profound vasodilation in the surrounding area (flare reaction, $P<0.01$). This response was specific and not induced by saline, albumin, acetylcholine or an ET-antagonist. Prolonged capsaicin pretreatment inhibited endothelin-1 induced vasodilation in the area surrounding the injection site, but not the central vasoconstriction at the injection site. Bradykinin also induced a marked vasodilation in the area surrounding the injection site; this was not inhibited by an ETA-selective antagonist, while the flare reaction was. **L-NMMA** applied at the site of the flare reaction prevented endothelin-1-induced vasodilation. CONCLUSIONS: Endothelin-1 in the human skin microcirculation stimulates polimodal nociceptor fibres leading to the release of **nitric oxide**. This response may play a pathophysiological role in inflammatory processes in the human skin.
CT Check Tags: Human; Male; Support, Non-U.S. Gov't
Adolescent
Adult
Capsaicin: PD, pharmacology
Endothelin-1: AI, antagonists & inhibitors
*Endothelin-1: PD, pharmacology
Enzyme Inhibitors: PD, pharmacology
Microcirculation: DE, drug effects
Nitric Oxide: ME, metabolism
*Nociceptors: DE, drug effects
Receptors, Endothelin: AI, antagonists & inhibitors
Receptors, Endothelin: PH, physiology
Skin: BS, blood supply
*Skin: DE, drug effects
Skin: ME, metabolism

*Vasodilation: DE, drug effects
omega-N-Methylarginine: PD, pharmacology
RN **10102-43-9 (Nitric Oxide)**; **17035-90-4 (omega-N-Methylarginine)**; 404-86-4 (Capsaicin)
CN 0 (Endothelin-1); 0 (Enzyme Inhibitors); 0 (PD 147953); 0 (Receptors, Endothelin)

L107 ANSWER 37 OF 47 MEDLINE on STN
AN 96236108 MEDLINE
DN PubMed ID: 9053788
TI Acute thermal hyperalgesia in the rat is produced by activation of N-methyl-D-aspartate receptors and protein kinase C and production of **nitric oxide.**
AU Meller S T; Dykstra C; Gebhart G F
CS Department of Pharmacology, University of Iowa, Iowa City 52242, USA.
NC DA 02879 (NIDA)
NS 29844 (NINDS)
SO Neuroscience, (1996 Mar) 71 (2) 327-35.
Journal code: 7605074. ISSN: 0306-4522.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199703
ED Entered STN: 19970321
Last Updated on STN: 19970321
Entered Medline: 19970313
AB There is general agreement that activation of the N-methyl-D-aspartate receptor is involved in thermal hyperalgesia. However, there is less agreement on the specific intracellular events subsequent to receptor activation and the involvement of other excitatory amino acid receptors in thermal hyperalgesia. In the present study, we found that the intrathecal administration of N-methyl-D-aspartate produced a dose- (1 fmol-1 pmol) and time-dependent thermal hyperalgesia. In contrast, over the dose range tested, intrathecal administration of either alpha-amino-3-hydroxy-5-methylisoxazole-4-proprionate (AMPA; 10 fmol-100 pmol), 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (10 fmol-100 pmol), quisqualate (10 pmol-5 nmol) or a 1:1 combination of AMPA and 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (total dose 20 fmol-200 pmol) did not produce any evidence of thermal hyperalgesia; greater doses produced a caudally-directed biting and **scratching** behavior that precluded testing in the paradigm used. A fixed dose of 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (100 pmol) did, however, potentiate the effects of N-methyl-D-aspartate (1-100 fmol). Thermal hyperalgesia produced by N-methyl-D-aspartate (1 pmol) was attenuated by intrathecal administration of the N-methyl-D-aspartate receptor-selective antagonist 2-amino-5-phosphonopentanoate (100 pmol), but not by the AMPA receptor-selective antagonist 6,7-dinitroquinoxaline-2,3-dione (1 nmol) or the metabotropic receptor antagonist 2-amino-3-phosphonoproprionate (10 nmol). In a second series of experiments, we examined the role of different signal transduction systems in acute N-methyl-D-aspartate-produced thermal hyperalgesia. N-Methyl-D-aspartate-produced thermal hyperalgesia (1 pmol) was attenuated by intrathecal hemoglobin (1-100 pmol) and dose-dependently by intrathecal N(G)-**nitro-L-arginine** methyl ester (10 pmol-1 nmol), Methylene Blue (10 pmol-1 nmol) and chelerythrine (1-100 pmol), suggesting that acute N-methyl-D-aspartate-mediated thermal hyperalgesia involves activation of **nitric oxide** synthase and protein kinase C. In contrast, N-methyl-D-aspartate-produced thermal hyperalgesia was unaffected by intrathecal administration of the phospholipase A2 inhibitor

mepacrine (10 nmol) or the phospholipase C inhibitor neomycin (10 nmol). While prostaglandins and leukotrienes have been suggested to play a role in hyperalgesia, N-methyl-D-aspartate-produced thermal hyperalgesia (1 pmol) was unaffected by the non-selective eicosanoid inhibitor nordihydroguaiarate (1 nmol), the cyclo-oxygenase selective inhibitor indomethacin (10 nmol) or the lipoxygenase selective inhibitor baicalein (1 nmol). The results of the present study suggest that acute thermal hyperalgesia can be produced by activation of N-methyl-D-aspartate receptors. Activation of AMPA, metabotropic or co-activation of AMPA and metabotropic glutamate receptors, at the doses tested, did not produce an acute thermal hyperalgesia. The thermal hyperalgesia produced by N-methyl-D-aspartate is mediated by activation of **nitric oxide** synthase and protein kinase C, but not by phospholipase C, phospholipase A2, cyclo-oxygenase or lipoxygenase. Collectively, the results are consistent with a role for spinal N-methyl-D-aspartate receptors, **nitric oxide** and protein kinase C in thermal hyperalgesia.

CT Check Tags: Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Animals
Biotransformation: DE, drug effects
Enzyme Activation: DE, drug effects
Excitatory Amino Acid Agonists: PD, pharmacology
Heat
Hyperalgesia: ME, metabolism
*Hyperalgesia: PP, physiopathology
Injections, Spinal
N-Methylaspartate: PD, pharmacology
***Nitric Oxide: BI, biosynthesis**
Pain Measurement: DE, drug effects
*Protein Kinase C: ME, metabolism
Rats
Rats, Sprague-Dawley
Receptors, AMPA: AG, agonists
*Receptors, N-Methyl-D-Aspartate: AG, agonists

RN **10102-43-9 (Nitric Oxide)**; 6384-92-5 (N-Methylaspartate)

CN 0 (Excitatory Amino Acid Agonists); 0 (Receptors, AMPA); 0 (Receptors, N-Methyl-D-Aspartate); EC 2.7.1.37 (Protein Kinase C)

=> b embase

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=> d all l148 1 3 6 8 10 16 17 22 29 31 34

L148 ANSWER 1 OF 44 EMBASE
on STN

AN 2001333593 EMBASE

TI Interaction of Bartonella henselae with the murine macrophage cell line J774: Infection and proinflammatory response.

AU Musso T.; Badolato R.; Ravarino D.; Stornello S.; Panzanelli P.; Merlino

C.; Savoia D.; Cavallo R.; Negro Ponzi A.; Zucca M.
CS T. Musso, Istituto di Microbiologia, Via Santena 9, 10126 Torino, Italy. tiziana.musso@unito.it
SO Infection and Immunity, (**2001**) 69/10 (5974-5980).
Refs: 40
ISSN: 0019-9567 CODEN: INFIBR
CY United States
DT Journal; Article
FS 004 Microbiology
026 Immunology, Serology and Transplantation
LA English
SL English
AB Bartonella henselae is the causative agent of cat **scratch disease** (CSD), a self-limiting condition characterized by a subacute regional lymphadenopathy that may develop into disseminated bartonellosis in immunocompromised subjects. Mice experimentally infected with B. henselae display typical liver and spleen granulomas rich in T cells and macrophages. So far there are no data on the interaction between bartonellae and macrophages. In order to clarify this topic, we investigated the interaction of B. henselae with J774, a mouse macrophage cell line. Analysis of bacterial uptake by functional assays and transmission electron microscopy indicates that bartonellae can enter and survive inside J774. Entry occurred within 30 min postinfection and reached a plateau at 160 min. Infection of J774 was followed by a dose-dependent release of the proinflammatory cytokines tumor necrosis factor alpha, interleukin 1β (IL-1β), and IL-6. Bartonellae persisted intracellularly without loss of viability for at least 8 h, and their number slightly decreased 24 h postinfection. Gamma interferon (IFN-γ) treatment of J774 significantly decreased the number of recoverable bacteria at 8 and 24 h. This enhancement of macrophage bactericidal activity was associated with **nitric oxide** (NO) release and was prevented by the addition of the competitive inhibitor of NO synthesis N(G)-monomethyl L-arginine. These findings suggest that IFN-γ-mediated activation of macrophages may be important for the clearing of B. henselae infection and that anti-B, henselae microbicidal activity of IFN-γ-activated macrophages is mediated to a large extent by NO production.
CT Medical Descriptors:
*Bartonella henselae
*rickettsiosis
*macrophage
cat scratch disease: ET, etiology
lymphadenopathy: CO, complication
immune deficiency
liver granuloma
spleen
transmission electron microscopy
dose response
cytokine release
bactericidal activity
macrophage activation
nonhuman
mouse
animal experiment
animal model
controlled study
animal cell
article
priority journal
Drug Descriptors:

cytokine: EC, endogenous compound
tumor necrosis factor alpha: EC, endogenous compound
interleukin 1beta: EC, endogenous compound
interleukin 6: EC, endogenous compound
nitric oxide
n(g) methylarginine
gamma interferon
RN (**nitric oxide**) **10102-43-9**; (n(g) **methylarginine**) **156706-47-7**, **17035-90-4**; (gamma interferon) 82115-62-6

L148 ANSWER 3 OF 44 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2000434392 EMBASE
TI The neurogenic vasodilator response to endothelin-1: A study in human skin in vitro.
AU Katugampola R.; Church M.K.; Clough G.F.
CS G.F. Clough, Allergy and Inflammation Sciences, School of Medicine, University of Southampton, Southampton, United Kingdom. G.F.Clough@soton.ac.uk
SO Experimental Physiology, (**2000**) 85/6 (839-846).
Refs: 36
ISSN: 0958-0670 CODEN: EXPHEZ
CY United Kingdom
DT Journal; Article
FS 002 Physiology
013 Dermatology and Venereology
LA English
SL English
AB We have investigated the mediators and mechanisms underlying the vasodilator effects of the potent vasoactive peptide, endothelin-1 (ET-1) and its isomers ET-2 and ET-3 in human skin, in vivo, using cutaneous microdialysis to quantify the release of mediators within the dermal response and scanning laser Doppler imaging to measure changes in blood flux. The effects of local anaesthesia, inhibition of **nitric oxide** synthase (NOS) by **L-NAME** and ET receptor blockade on the ET-induced vascular response were also investigated. ET-1, -2 and -3 all caused a dose-dependent area of pallor surrounded by a long-lasting flare which was accompanied by a short-lived burning **pruritus.** The concentration of **nitric oxide** (NO) in dialysate collected within the pallor response to 5 μm ET-1 (1·43 ± 0·64 μm, n = 5) was not significantly different from baseline levels collected prior to injection (0·86 ± 0·38 μm) whilst that in the flare increased to reach a peak value of 2·28 ± 0·61 μm at between 4 and 10 min after intradermal injection ($P < 0·004$). Pretreatment with local anaesthetic slowed the development of the flare and significantly reduced its size by up to 52% at 20 min after injection ($P < 0·05$) but had no significant effect on the central pallor. **L-NAME**, delivered by dialysis also caused a significant reduction in the ET-1-induced flare ($P < 0·005$). Bosentan, the non-selective ET(A)/ET(B) antagonist, when given by dialysis at the site of injection, reduced the area of both the ET-1-induced pallor and surrounding flare by 41 and 26%, respectively. No significant increase in tissue histamine was measured within either the pallor or flare response to ET-1, -2 or -3. Together these data confirm that the vasodilator response to endothelin-1 in human skin is neurogenic in origin and that it is in part mediated by the local release of **nitric oxide.** There appears to be little evidence for the involvement of mast cell-derived histamine in the initiation or modulation of ET-induced vasodilatation, in vivo.

CT Medical Descriptors:
in vivo study
skin blood vessel
vasodilatation
microdialysis
laser Doppler flowmetry
skin blood flow
local anesthesia
enzyme inhibition
pallor
skin manifestation
pruritus
dialysate
histamine release
mast cell
human
human experiment
normal human
controlled study
adult
article
Drug Descriptors:
*endothelin 1: EC, endogenous compound
local anesthetic agent
nitric oxide synthase inhibitor
n(g) nitroarginine methyl ester
bosentan
nitric oxide synthase: EC, endogenous compound
endothelin receptor antagonist
endothelin 2: EC, endogenous compound
endothelin 3: EC, endogenous compound
nitric oxide: EC, endogenous compound
histamine: EC, endogenous compound
endothelin receptor: EC, endogenous compound
RN (n(g) **nitroarginine** methyl ester) 50903-99-6; (**nitric oxide** synthase) 125978-95-2; (**nitric oxide**) **10102-43-9**; (histamine) 51-45-6, 56-92-8, 93443-21-1

L148 ANSWER 6 OF 44 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2000147443 EMBASE
TI The role of **nitric oxide** and prostaglandin E2 on the hyperalgesia induced by excitatory amino acids in rats.
AU Park Y.H.; Shin C.Y.; Lee T.S.; Huh I.H.; Sohn U.D.
CS U.D. Sohn, Department of Pharmacology, School of Pharmacy, Chung Ang University, 221 Huksuk-dong, Dongjak-Ku, Seoul 156-756, Korea, Republic of. udsohn@cau.ac.kr
SO Journal of Pharmacy and Pharmacology, (**2000**) 52/4 (431-436).
Refs: 28
ISSN: 0022-3573 CODEN: JPPMAB
CY United Kingdom
DT Journal; Article
FS 030 Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LA English
SL English
AB The present study was designed to investigate the role of **nitric oxide** (NO), N-methyl-D-aspartate (NMDA) receptor and prostaglandins on hyperalgesia induced in rats by excitatory amino acids

and the possibility that prostaglandins may act as the retrograde messenger in the spinal cord like NO. **Nω - nitro-L-arginine** methyl ester (**L-NAME**; 500 μg/paw, intraplantarly (i.pl.)), MK-801 (10 μg/paw, i.pl.) or indomethacin (300 μg/paw, i.pl.) reduced the duration of phase 2 of the biting/licking and **scratching** (B/L + S) response induced by formalin injection from 255.6±16.7 s to 155.6±16.9, 172.2±33.3 or 205.6±16.7 s, respectively. **L-NAME** (0.3 mg, i.th.), MK-801 (8 μg, i.th.) or indomethacin (20 μg, i.th.) reduced the duration of phase 2 of the B/L + S response induced by saline injection from 288.5±7.7 s to 207.7±19.2, 184.6±7.7 or 192.3±38.5 s, respectively. **L-NAME** or indomethacin injected into the spinal cord of the rat significantly reduced the hyperalgesia induced by NMDA (1 μg, i.th.) from 43.8±4.6% to 12.3±3.1 and 19.2±2.3%, respectively. It is assumed that NO produced by excitatory amino acids may increase prostaglandin production by cyclooxygenase activation. **L-NAME**, MK-801 or indomethacin injected into the rat spinal cord significantly reduced the hyperalgesia induced by prostaglandin E2 (PGE2, 25 ng, i.th.) in the tail-flick test from 40.6±3.5% to 18.2±3.2, 18.8±1.8 or 17.6±4.1%, respectively, but had little effect on hyperalgesia in the paw pressure test (except for indomethacin). In conclusion, NO and PGE2 affect the hyperalgesia induced by excitatory amino acids. It is suggested that PGE2, like NO, may act as a retrograde messenger in the spinal cord.

CT Medical Descriptors:
*hyperalgesia
nonhuman
animal experiment
rat
male
controlled study
drug effect
intraplantar drug administration
licking
scratching
bite
article
Drug Descriptors:
***nitric oxide: EC, endogenous compound**
*prostaglandin E2: PD, pharmacology
*n methyl dextro aspartic acid receptor: EC, endogenous compound
*arginine methyl ester: PD, pharmacology
*arginine methyl ester: AD, drug administration
*indometacin: PD, pharmacology
*n methyl dextro aspartic acid: PD, pharmacology
*n methyl dextro aspartic acid: TL, intrathecal drug administration
*dizocilpine: PD, pharmacology
*dizocilpine: AD, drug administration

RN (**nitric oxide**) **10102-43-9**; (prostaglandin E2) 363-24-6; (arginine methyl ester) 2577-94-8; (indometacin) 53-86-1, 74252-25-8, 7681-54-1; (n methyl dextro aspartic acid) 6384-92-5; (dizocilpine) 77086-21-6

CO Sigma (United States)

L148 ANSWER 8 OF 44 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AN 1999401721 EMBASE

TI Involvement of **nitric oxide** in **itch-scratch** response of NC mice.

AU Tsukumo Y.; Andoh T.; Yamaguchi T.; Nojima H.; Kuraishi Y.

CS Y. Tsukumo, Department of Applied Pharmacology, Faculty of Pharmaceutical Sciences, Toyama Med. and Pharmaceut. Univ., 2630 Sugitani, Toyama 930-0194, Japan
SO Folia Pharmacologica Japonica, (**1999**) 114/SUPPL. 1 (17P-21P).
Refs: 9
ISSN: 0015-5691 CODEN: NYKZAU
CY Japan
DT Journal; Article
FS 013 Dermatology and Venereology
037 Drug Literature Index
LA Japanese
SL English; Japanese
AB NC mice, a model for atopic dermatitis, showed **scratching** behavior when kept under conventional environment. The **scratching** behavior of NC mice was suppressed by distraction or by the administration of naltrexone (1 mg/kg, s.c.), an opioid antagonist. These results suggest that such **scratching** behavior is **itch**-associated response. The **itch**-associated response of the NC mice was significantly suppressed by an intravenous injection of **nitric oxide** (NO) synthase inhibitor N(G)-**nitro**-**L**-**arginine** methyl ester (**L**-**NAME**, 10 mg/kg), but not D-**NAME** (10 mg/kg) and saline. Intracutaneous NO production in the rostral back, a region which the NC mice mainly **scratched** , was markedly increased as compared with thc caudal back, a non-**scratched** region. The increased NO production in the rostral back of NC mice was decreased by the intravenous injection of **L**-**NAME** (10 mg/kg). These results suggest that NO and NO sythase are now target in the treatment of atopic **pruritus.**
CT Medical Descriptors:
***pruritus: DT, drug therapy**
***scratching**
*atopic dermatitis
microdialysis
nonhuman
mouse
animal experiment
animal model
controlled study
oral drug administration
subcutaneous drug administration
intravenous drug administration
article
Drug Descriptors:
***nitric oxide**
*naltrexone: DT, drug therapy
***n(g) nitroarginine methyl ester: DT, drug therapy**
n(g) nitro dextro arginine methyl ester
RN (**nitric oxide**) **10102-43-9**; (naltrexone) 16590-41-3, 16676-29-2; (n(g) **nitroarginine** methyl ester) 50903-99-6

L148 ANSWER 10 OF 44 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 1999274687 EMBASE
TI Nociceptin-induced **scratching**, biting and licking in mice: Involvement of spinal NK1 receptors.
AU Sakurada T.; Katsuyama S.; Sakurada S.; Inoue M.; Tan-No K.; Kisara K.; Sakurada C.; Ueda H.; Sasaki J.
CS T. Sakurada, Department of Biochemistry, Daiichi College Pharmaceutical Sci., 22-1 Tamagawa-cho, Minami-ku, Fukuoka 815-8511, Japan

SO British Journal of Pharmacology, (**1999**) 127/7 (1712-1718).
Refs: 38
ISSN: 0007-1188 CODEN: BJPCBM
CY United Kingdom
DT Journal; Article
FS 030 Pharmacology
037 Drug Literature Index
LA English
SL English

AB 1. Intrathecal (i.t.) injection of nociceptin at small doses (fmol order) elicited a behavioural response consisting of **scratching**, biting and licking in conscious mice. Here we have examined the involvement of substance P-containing neurons by using i.t. injection of tachykinin neurokinin (NK)1 receptor antagonists and substance P (SP) antiserum. 2. Nociceptin-induced behavioural response was evoked significantly 5-10 min after i.t. injection and reached a maximum at 10-15 min. Dose-dependency of the induced response showed a bell-shaped pattern from 0.375-30.0 fmol, and the maximum effect was observed at 3.0 fmol. 3. The behavioural response elicited by nociceptin (3.0 fmol) was dose-dependently inhibited by intraperitoneal (i.p.) administration of morphine. 4. The NK1 receptor antagonists, CP-96,345, CP-99,994 and sendide, inhibited nociceptin-induced behavioural response in a dose-dependent manner. A significant antagonistic effect of [D-Phe7, D-His9]SP (6-11), a selective antagonist for SP receptors, was observed against nociceptin-induced response. The NK2 receptor antagonist, MEN-10376, had no effect on the response elicited by nociceptin. 5. Pretreatment with SP antiserum resulted in a significant reduction of the response to nociceptin. No significant reduction of nociceptin-induced response was detected in mice pretreated with NKA antiserum. 6. The N-methyl-D-aspartate (NMDA) receptor antagonists, dizocilpine (MK-801) and D(-)-2-amino5-phosphonovaleric acid (APV) (D-APV), and L-N(G)-nitro arginine methyl ester (**L-NAME**), a **nitric oxide** (NO) synthase inhibitor, failed to inhibit nociceptin-induced behavioural response. 7. The present results suggest that SP-containing neurons in the mouse spinal cord may be involved in elicitation of **scratching**, biting and licking behaviour following i.t. injection of nociceptin.

CT Medical Descriptors:
licking
scratching
bite
nonhuman
male
mouse
animal experiment
intrathecal drug administration
article
priority journal
Drug Descriptors:
*neurokinin 1 receptor: EC, endogenous compound
*nociceptin: PD, pharmacology
neuropeptide: PD, pharmacology
substance p: EC, endogenous compound
2 benzhydryl 3 (2 methoxybenzylamino) 1 azabicyclo[2.2.2]octane: PD, pharmacology
3 (2 methoxybenzylamino) 2 phenylpiperidine: PD, pharmacology
neurokinin a [4-10][5 tyrosine 6,8,9 dextro tryptophan 10 lysine]: PD, pharmacology
tachykinin receptor antagonist: PD, pharmacology
substance p antibody: PD, pharmacology
n methyl dextro aspartic acid receptor blocking agent: PD, pharmacology

dizocilpine: PD, pharmacology
2 amino 5 phosphonovaleric acid: PD, pharmacology
arginine methyl ester: PD, pharmacology
nitric oxide synthase inhibitor: PD, pharmacology
RN (nociceptin) 170713-75-4; (substance p) 33507-63-0; (2 benzhydryl 3 (2 methoxybenzylamino) 1 azabicyclo[2.2.2]octane) 132746-60-2, 134731-58-1; (3 (2 methoxybenzylamino) 2 phenylpiperidine) 136982-36-0; (neurokinin a [4-10] [5 tyrosine 6,8,9 dextro tryptophan 10 lysine]) 135306-85-3; (dizocilpine) 77086-21-6; (2 amino 5 phosphonovaleric acid) 76726-92-6; (arginine methyl ester) 2577-94-8
CN (1) Men 10376; (2) Mk 801; (3) Cp 96345; (4) Cp 99994
CO (1) Sankyo (Japan); (2) Research Biochemicals (United States); (4) Pfizer; Cambridge Research (United Kingdom); Wako (Japan)

L148 ANSWER 16 OF 44 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 1998192700 EMBASE
TI Basal **nitric oxide** release differentially modulates vasodilations by pinacidil and levcromakalim in goat coronary artery.
AU Deka D.K.; Raviprakash V.; Mishra S.K.
CS S.K. Mishra, Div. of Pharmacology and Toxicology, Indian Veterinary Research Institute, Izatnagar-243 11, India. wood.michael@mayo.edu
SO European Journal of Pharmacology, (**1 May 1998**) 348/1 (11-23).
Refs: 36
ISSN: 0014-2999 CODEN: EJPHAZ
PUI S 0014-2999(98)00066-1
CY Netherlands
DT Journal; Article
FS 030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB In the current investigation, the role of basal **nitric oxide** (NO) in modulating the vasorelaxant responses to pinacidil and levcromakalim was examined in goat isolated coronary artery. Pinacidil (10-8 - 10-4 M) elicited concentration-dependent relaxations of the coronary artery ring segments (with intact endothelium constricted with 30 mM K+ saline solution. The EC50 of the vasodilator was 2.57 x 10-6 M (95% CL, 1.9-3.46 x 10-6 M). The removal of endothelium by mechanical **rubbing** caused a rightward shift in the concentration-response curve of pinacidil with a corresponding increase in EC50 value (1.90 x 10-5 M; 95% CL, 1.12-3.23 x 10-5 M). Similar to endothelium removal, treatment of endothelium-intact rings either with the NO synthesis inhibitor **L-NAME** (N(G)-**nitro-L-arginine** methyl ester; 3 x 10-5 M) or the guanylate cyclase inhibitor, methylene blue (3 x 10-6 M) resulted in a marked inhibition in the relaxant responses to pinacidil. Hence, the EC50 values of the potassium channel opener were significantly higher in tissues treated either with **L-NAME** (7.41 x 10-6 M; 95% CL, 6.02- 9.12 x 10-6 M) or methylene blue (2.29 x 10-5 M; 95% CL, 1.5 ± -3.31 x 10-5 M) as compared to untreated controls. The ATP-sensitive potassium (K(ATP)) channel blocker glibenclamide, which caused a significant rightward shift in the concentration-relaxation curve of pinacidil in control tissues, was found to be less potent in antagonising the relaxant responses of the K(ATP) channel opener in endothelium-denuded rings and in rings with intact endothelium but treated with either **L-NAME** or methylene blue. In contrast to the observations made with pinacidil, the vasodilator responses to another K(ATP) channel opener, levcromakalim, were potentiated in the absence of basal NO. Thus, the EC50 of levcromakalim was 1.33 x 10-8 M (95% CL, 0.8- 2.21 x 10-8 M) in control

tissues with intact endothelium, which was significantly higher than those obtained in endothelium-deprived rings (4.81 x 10-9 M; 95% CL, 4.04-5.73 x 10-9 M) or endothelium intact rings treated either with **L-NAME** (2.63 x 10-9 M; 95% CL, 1.58-4.36 x 10-9 M) or methylene blue (2.82 x 10-9 M; 95% CL, 1.7-4.68 x 10-9 M). The detective modulation by basal NO of the arterial relaxations elicited with the K(ATP channel openers was evident from the findings that papaverine-induced relations were not affected in the absence of basal NO. Taken together, the results of the present study suggest that basal NO differentially modulates the interaction of pinacidil and levcromakalim with the K(ATP) channels in goat coronary artery through a cGMP-dependent pathway.

CT Medical Descriptors:
*coronary artery dilatation
goat
concentration response
drug potency
drug mechanism
potassium channel
artery endothelium
drug effect
drug antagonism
nonhuman
controlled study
animal tissue
article
priority journal
Drug Descriptors:
*potassium channel stimulating agent: PD, pharmacology
*pinacidil: PD, pharmacology
*papaverine: PD, pharmacology
*potassium channel blocking agent: PD, pharmacology
*glibenclamide: PD, pharmacology
***nitric oxide: EC, endogenous compound**
n(g) nitroarginine methyl ester: PD, pharmacology
methylene blue: PD, pharmacology
indometacin: PD, pharmacology
linsidomine: PD, pharmacology
lemakalim: PD, pharmacology
clonidine: PD, pharmacology
propranolol
prazosin
noradrenalin

RN (pinacidil) 60560-33-0; (papaverine) 58-74-2, 61-25-6; (glibenclamide) 10238-21-8; (**nitric oxide**) **10102-43-9**; (n(g) **nitroarginine** methyl ester) 50903-99-6; (methylene blue) 61-73-4; (indometacin) 53-86-1, 74252-25-8, 7681-54-1; (linsidomine) 16142-27-1, 33876-97-0; (lemakalim) 94535-50-9; (clonidine) 4205-90-7, 4205-91-8, 57066-25-8; (propranolol) 13013-17-7, 318-98-9, 3506-09-0, 4199-09-1, 525-66-6; (prazosin) 19216-56-9, 19237-84-4; (noradrenalin) 1407-84-7, 51-41-2

CO Hoechst (Germany); Sigma; Ingelheim pharmaceuticals (Germany); Leo pharmaceuticals (Denmark); Smith kline beecham (United Kingdom); Pfizer; May and baker

L148 ANSWER 17 OF 44 EMBASE
on STN
AN 1998166238 EMBASE
TI Endothelin-1 induces vasodilation in human skin by nociceptor fibres and release of **nitric oxide.**
AU Wenzel R.R.; Zbinden S.; Noll G.; Meier B.; Luscher T.F.

CS Prof. T.F. Luscher, Department of Cardiology, University Hospital, CH-8091 Zurich, Switzerland
SO British Journal of Clinical Pharmacology, (**1998**) 45/5 (441-446).
Refs: 37
ISSN: 0306-5251 CODEN: BCPHBM
CY United Kingdom
DT Journal; Article
FS 002 Physiology
018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB Aims - Endothelin is a peptide produced by endothelial cells with many biological properties. In the human skin microcirculation endothelin induces neurogenic vasodilation associated with burning **pruritus**. We investigated the mechanisms involved in this response. Methods - The effects of prolonged pretreatment with capsaicin, a specific inhibitor of polimodal nociceptor fibres, and of the **nitric oxide** synthase inhibitor **L-NMMA** on endothelin-1-induced vasodilation were studied in 15 human subjects. Furthermore, we investigated the effects of the ET(A)-selective antagonist PD147953 on bradykinin-induced vasodilation. Results - After local injection, endothelin-1 caused vasoconstriction at the injection site and a profound vasodilation in the surrounding area (flare reaction, $P < 0.01$). This response was specific and not induced by saline, albumin, acetylcholine or an ET-antagonist. Prolonged capsaicin pretreatment inhibited endothelin-1 induced vasodilation in the area surrounding the injection site, but not the central vasoconstriction at the injection site. Bradykinin also induced a marked vasodilation in the area surrounding the injection site; this was not inhibited by an ETA-selective antagonist, while the flare reaction was. **L-NMMA** applied at the site of the flare reaction prevented endothelin-1-induced vasodilation. Conclusions - Endothelin-1 in the human skin microcirculation stimulates polimodal nociceptor fibres leading to the release of **nitric oxide**. This response may play a pathophysiological role in inflammatory processes in the human skin.
CT Medical Descriptors:
*vasodilatation
*nociceptive receptor
*skin
*vasoconstriction
human
male
human experiment
normal human
adult
article
priority journal
Drug Descriptors:
*endothelin 1: PD, pharmacology
***nitric oxide: EC, endogenous compound**
*capsaicin: PD, pharmacology
*pd 147953: DV, drug development
*pd 147953: PD, pharmacology
*endothelin receptor antagonist: DV, drug development
*endothelin receptor antagonist: PD, pharmacology
*noradrenalin: PD, pharmacology
***n(g) methylarginine: PD, pharmacology**

bradykinin
nitric oxide synthase inhibitor: PD, pharmacology
acetylcholine: PD, pharmacology
unclassified drug
RN (**nitric oxide**) **10102-43-9**; (capsaicin) 404-86-4; (noradrenalin) 1407-84-7, 51-41-2; (n(g) **methylarginine**) **17035-90-4**; (bradykinin) 58-82-2, 5979-11-3; (acetylcholine) 51-84-3, 60-31-1, 66-23-9
CN (1) Pd 147953
CO (1) Parke davis; Clinalfa

L148 ANSWER 22 OF 44 EMBASE
on STN
AN 97249574 EMBASE
DN 1997249574
TI **Nitric oxide**-mediated vasorelaxation induced by sodium polyoxyethylene laurylether sulfate.
AU Koyama K.; Kasuya Y.; Koyama K.; Goto K.
CS K. Koyama, Department of Emergency Medicine, Institute of Clinical Medicine, University of Tsukuba, 1-1-1 Tennoudai, Tsukuba, Ibaraki 305, Japan
SO Toxicology and Applied Pharmacology, (**1997**) 145/2 (294-300).
Refs: 36
ISSN: 0041-008X CODEN: TXAPA
CY United States
DT Journal; Article
FS 002 Physiology
037 Drug Literature Index
LA English
SL English
AB Ingestion of surfactants is known to cause hemodynamic changes with decreased total vascular resistance. Motivated by this clinical observation, we investigated the direct effects of a common anionic surfactant, sodium polyoxyethylene laurylether sulfate (LES), on isolated ring segments of rat thoracic aorta. LES did not produce any vasocontractile responses, but relaxed ring segments precontracted with 10-6 M phenylephrine in a concentration-dependent manner. This LES-induced vasorelaxation was significantly reduced by the removal of endothelium or pretreatment with N(G)-**nitro-L-arginine** methylester hydrochloride, methylene blue, or oxyhemoglobin to the same degree, but was not affected by pretreatment with indomethacin. A further study measuring NO2- plus NO3- (NO(x), total metabolites of NO) in the medium of calf pulmonary artery endothelial (CPAE) cells, a cultured cell line, revealed that LES caused a significant increase in NO(x) production. On the other hand, in a study measuring intracellular Ca2+ in fura-2-loaded CPAE cells, LES caused a significant increase in intracellular Ca2+. These results suggest that LES causes endothelium-dependent vasorelaxation via a NO-mediated signaling pathway, which might be due to Ca2+ mobilization.
CT Medical Descriptors:
*thoracic aorta
*vasodilatation
animal experiment
animal tissue
article
calcium mobilization
controlled study
drug effect
endothelium cell
hemodynamics

male
nonhuman
Drug Descriptors:
***nitric oxide**
***polidocanol: PD, pharmacology**
indometacin
methylene blue
n(g) nitroarginine methyl ester
oxyhemoglobin
phenylephrine
surfactant
RN (**nitric oxide**) **10102-43-9**; (polidocanol) 60828-78-6, 9002-92-0; (indometacin) 53-86-1, 74252-25-8, 7681-54-1; (methylene blue) 61-73-4; (n(g) **nitroarginine** methyl ester) 50903-99-6; (oxyhemoglobin) 9061-63-6; (phenylephrine) 532-38-7, 59-42-7, 61-76-7
CO Wako (Japan)

L148 ANSWER 29 OF 44 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 96249258 EMBASE
DN 1996249258
TI Involvement of **nitric oxide** in spinally mediated capsaicin- and glutamate-induced behavioural responses in the mouse.
AU Sakurada T.; Sugiyama A.; Sakurada C.; Tanno K.; Sakurada S.; Kisara K.; Hara A.; Abiko Y.
CS Department of Biochemistry, Daiichi College Pharmaceutical Sci., 22-1 Tamagawa-cho,Minami-ku, Fukuoka 815, Japan
SO Neurochemistry International, (**1996**) 29/3 (271-278).
ISSN: 0197-0186 CODEN: NEUIDS
CY United Kingdom
DT Journal; Article
FS 002 Physiology
008 Neurology and Neurosurgery
LA English
SL English
AB The intrathecal (i.t.) injection of capsaicin (0.1 nmol/mouse) through a lumbar puncture elicited **scratching**, biting and licking responses. Pretreatment with the **nitric oxide** synthase inhibitor N(G)-**nitro**-**L**-**arginine** methyl ester (**L**-**NAME**) (320 nmol), by i.t. injection, resulted in a significant inhibition of the behavioural response produced by i.t. capsaicin (0.1 nmol/mouse). Similar behavioural responses were induced by i.t. injections of NMDA (0.4 nmol), kainate (0.05 nmol) or AMPA (0.05 nmol), which were all inhibited by co-administration of **L**-**NAME** (20-80 nmol). L-Arginine (600 mg/kg, i.p.) but not Darginine (600 mg/kg, i.p.) reversed the inhibitory effect of **L**-**NAME** on capsaicin-, NMDA-, kainate- and AMPA-induced behavioural response. **Scratching**, biting and licking responses induced by tachykinin receptor agonists, substance P, [Sar9,Met(O2)11]substance P, neurokinin A and neurokinin B were not affected by co-administration of **L**-**NAME** (40 and 80 nmol). These results suggest that spinal **nitric oxide** may play a significant role in mechanisms of the behavioural response to capsaicin, probably through the release of glutamate, but not tachykinins.
CT Medical Descriptors:
*bite
*licking
***scratching**
animal behavior

animal experiment
article
controlled study
lumbar puncture
male
mouse
neurotransmitter release
nonhuman
priority journal
spinal cord
Drug Descriptors:
*capsaicin
*glutamic acid
***nitric oxide: EC, endogenous compound**
alpha amino 3 hydroxy 5 methyl 4 isoxazolepropionic acid
arginine
dextro arginine
kainic acid
n methyl dextro aspartic acid
n(g) nitroarginine methyl ester
neurokinin a
neurokinin b
substance p
substance p derivative
tachykinin receptor agonist

RN (capsaicin) 404-86-4; (glutamic acid) 11070-68-1, 138-15-8, 56-86-0, 6899-05-4; (**nitric oxide**) **10102-43-9**; (alpha amino 3 hydroxy 5 methyl 4 isoxazolepropionic acid) 77521-29-0; (arginine) 1119-34-2, 15595-35-4, 7004-12-8, 74-79-3; (kainic acid) 487-79-6; (n methyl dextro aspartic acid) 6384-92-5; (n(g) **nitroarginine** methyl ester) 50903-99-6; (neurokinin a) 86933-74-6; (neurokinin b) 86933-75-7; (substance p) 33507-63-0

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AN 96062226 EMBASE

DN 1996062226

TI Acute thermal hyperalgesia in the rat is produced by activation of N-methyl-D-aspartate receptors and protein kinase C and production of **nitric oxide.**

AU Meller S.T.; Dykstra C.; Gebhart G.F.

CS Department of Pharmacology, University of Iowa,Iowa City, IA 52242, United States

SO Neuroscience, (**1996**) 71/2 (327-335).
ISSN: 0306-4522 CODEN: NRSCDN

CY United Kingdom

DT Journal; Article

FS 002 Physiology
030 Pharmacology
037 Drug Literature Index

LA English

SL English

AB There is general agreement that activation of the N-methyl-D-aspartate receptor is involved in thermal hyperalgesia. However, there is less agreement on the specific intracellular events subsequent to receptor activation and the involvement of other excitatory amino acid receptors in thermal hyperalgesia. In the present study, we found that the intrathecal administration of N-methyl-D-aspartate produced a dose- (1 fmol-1 pmol) and time-dependent thermal hyperalgesia. In contrast, over the dose range tested intrathecal administration of either α-amino-3-hydroxy-5-

methylisoxazole-4-proprionate (AMPA; 10 fmol-100 pmol), 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (10 fmol-100 pmol), quisqualate (10 pmol-5 nmol) or a 1:1 combination of AMPA and 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (total dose 20 fmol-200 pmol) did not produce any evidence of thermal hyperalgesia, greater doses produced a caudally-directed biting and **scratching** behavior that precluded testing in the paradigm used. A fixed dose of 1,3-trans-1-aminocyclopentyl-1,3-dicarboxylate (100 pmol) did, however, potentiate the effects of N-methyl-D-aspartate (1-100 fmol). Thermal hyperalgesia produced by N-methyl-D-aspartate (1 pmol) was attenuated by intrathecal administration of the N-methyl-D-aspartate receptor-selective antagonist 2-amino-5-phosphonopentanoate (100 pmol), but not by the AMPA receptor-selective antagonist 6,7-dinitroquinoxaline-2,3-dione (1 nmol) or the metabotropic receptor antagonist 2-amino-3-phosphonoproprionate (10 nmol). In a second series of experiments, we examined the role of different signal transduction systems in acute N-methyl-D-aspartate-produced thermal hyperalgesia. N-Methyl-D-aspartate-produced thermal hyperalgesia (1 pmol) was attenuated by intrathecal hemoglobin (1-100 pmol) and dose-dependently by intrathecal N(G)-**nitro-L-arginine** methyl ester (10 pmol-1 nmol), Methylene Blue (10 pmol-1 nmol) and chelerythrine (1-100 pmol), suggesting that acute N-methyl-D-aspartate-mediated thermal hyperalgesia involves activation of **nitric oxide** synthase and protein kinase C. In contrast, N-methyl-D-aspartate-produced thermal hyperalgesia was unaffected by intrathecal administration of the phospholipase A2 inhibitor mepacrine (10 nmol) or the phospholipase C inhibitor neomycin (10 nmol). While prostaglandins and leukotrienes have been suggested to play a role in hyperalgesia, N-methyl-D-aspartate-produced thermal hyperalgesia (1 pmol) was unaffected by the non-selective eicosanoid inhibitor nordihydroguaiarate (1 nmol), the cyclo-oxygenase selective inhibitor indomethacin (10 nmol) or the lipoxygenase selective inhibitor baicalein (1 nmol). The results of the present study suggest that acute thermal hyperalgesia can be produced by activation of N-methyl-D-aspartate receptors. Activation of AMPA, metabotropic or co-activation of AMPA and metabotropic glutamate receptors, at the doses tested, did not produce an acute thermal hyperalgesia. The thermal hyperalgesia produced by N-methyl-D-aspartate is mediated by activation of **nitric oxide** synthase and protein kinase C, but not by phospholipase C, phospholipase A2, cyclo-oxygenase or lipoxygenase. Collectively, the results are consistent with a role for spinal N-methyl-D-aspartate receptors, **nitric oxide** and protein kinase C in thermal hyperalgesia.

CT Medical Descriptors:
*hyperalgesia
animal experiment
article
behavior
controlled study
heat
male
nonhuman
priority journal
rat
Drug Descriptors:
*2 amino 5 phosphonovaleric acid: PD, pharmacology
*alpha amino 3 hydroxy 5 methyl 4 isoxazolepropionic acid
*chelerythrine
*glutamic acid antagonist
*hemoglobin
*metabotropic receptor: EC, endogenous compound

*methylene blue
*n methyl dextro aspartic acid
*n methyl dextro aspartic acid receptor: EC, endogenous compound
***n(g) nitroarginine methyl ester**
*protein kinase c: EC, endogenous compound
*quisqualic acid
2 amino 3 phosphonopropionic acid
6,7 dinitro 2,3 quinoxalinedione
baicalein
indometacin
mepacrine
neomycin
nordihydroguaiaretic acid

RN (2 amino 5 phosphonovaleric acid) 76726-92-6; (alpha amino 3 hydroxy 5 methyl 4 isoxazolepropionic acid) 77521-29-0; (chelerythrine) 34316-15-9; (hemoglobin) 9008-02-0; (methylene blue) 61-73-4; (n methyl dextro aspartic acid) 6384-92-5; (n(g) **nitroarginine** methyl ester) 50903-99-6; (protein kinase c) 141436-78-4; (quisqualic acid) 52809-07-1; (2 amino 3 phosphonopropionic acid) 5652-28-8; (6,7 dinitro 2,3 quinoxalinedione) 2379-57-9; (baicalein) 491-67-8; (indometacin) 53-86-1, 74252-25-8, 7681-54-1; (mepacrine) 69-05-6, 83-89-6; (neomycin) 11004-65-2, 1404-04-2, 1405-10-3, 8026-22-0; (nordihydroguaiaretic acid) 500-38-9

CO Sigma

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AN 94301780 EMBASE

DN 1994301780

TI **Nitric oxide** and proteoglycan biosynthesis by human articular chondrocytes in alginate culture.

AU Hauselmann H.J.; Oppliger L.; Michel B.A.; Stefanovic-Racic M.; Evans C.H.

CS Department of Rheumatology, University Hospital, Gloriastrasse 25,8091 Zurich, Switzerland

SO FEBS Letters, (**1994**) 352/3 (361-364).
ISSN: 0014-5793 CODEN: FEBLAL

CY Netherlands

DT Journal; Article

FS 029 Clinical Biochemistry
031 Arthritis and Rheumatism
030 Pharmacology
037 Drug Literature Index

LA English

SL English

AB Interleukin-1α and β induced the production of large amounts of **nitric oxide** by normal, human articular chondrocytes in alginate culture; at the same time the biosynthesis of proteoglycan was strongly suppressed. In a dose-dependent manner, N(G)-monomethyl-L-arginine both inhibited **nitric oxide** formation and relieved the suppression of proteoglycan synthesis. However concentrations of N(G)-monomethyl-L-arginine which completely prevented **nitric oxide** production only partially restored proteoglycan biosynthesis, even at low doses of interleukin-1 where suppression of proteoglycan synthesis was modest. The organic donor of **nitric oxide**, S-nitrosyl-acetyl-D,L-penicillamine also inhibited proteoglycan biosynthesis, but not as extensively as interleukin-1. These data suggest that interleukin-1 suppresses synthesis of the cartilaginous matrix through more than one mechanism, at least one of which is dependent upon the production of **nitric oxide.**

CT Medical Descriptors:

*articular cartilage
*proteoglycan synthesis
adult
article
controlled study
human
human cell
normal human
priority journal
Drug Descriptors:
***alginic acid**
*interleukin 1: TO, drug toxicity
*interleukin 1: DO, drug dose
***n(g) methylarginine: PD, pharmacology**
***n(g) methylarginine: DO, drug dose**
***nitric oxide: EC, endogenous compound**
*proteoglycan: EC, endogenous compound
n acetyl s nitrosopenicillamine
recombinant alpha interferon beta: TO, drug toxicity
recombinant alpha interferon beta: DO, drug dose
recombinant interleukin 1 receptor blocking agent: PD, pharmacology
unclassified drug
RN (alginic acid) 28961-37-7, 29894-36-8, 9005-32-7, 9005-38-3; (n(g) **methylarginine) 17035-90-4; (nitric oxide) 10102-43-9;** (n acetyl s nitrosopenicillamine) 79032-48-7
CO Ciba geigy (Switzerland); Sigma (United States); Alexis corporation (Switzerland); R and d systems (United Kingdom)

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=> d all l200 tot

L200 ANSWER 1 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2003-748327 [70] WPIX
DNC C2003-205203
TI **Nitric oxide** synthase inhibitor selective for inducible form contains new or known optically active thiazole or oxazole compounds used for treating e.g. myocardial disease, Alzheimer's, septicemia, arthritis and gastric ulcer.
DC B03
IN IDO, M; MATSUMOTO, M; TERAUCHI, H; TSUJI, J; UEDA, S; YANO, A
PA (DAIN) DAINIPPON PHARM CO LTD
CYC 99
PI WO 2003075922 A1 20030918 (200370)* JA 39p A61K031-421
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW
ADT WO 2003075922 A1 WO 2002-JP2392 20020314
PRAI WO 2002-JP2392 20020314
IC ICM A61K031-421
ICS A61K031-426; A61P001-04; A61P003-10; A61P009-00; A61P009-02; A61P009-10; A61P019-02; A61P025-00; A61P025-28; A61P029-00; A61P043-00; C07D227-04; C07D263-28
AB WO2003075922 A UPAB: 20031030
NOVELTY - **Nitric oxide** synthase inhibitor contains optically active 4 and/or 5-alkyl-2-amino-oxazole or -thiazole (I).
DETAILED DESCRIPTION - **Nitric oxide** synthase inhibitor contains an aminooxazole or **aminothiazole** compound of formula (I), or its salts.
R1, R2 = H or lower alkyl, and
X = O or S,
provided that R1 and R2 are not both H.
An INDEPENDENT CLAIM is also included for new compounds (I: R1 and R2 = lower alkyl), provided that R1 and R2 are not both methyl when X = S.
ACTIVITY - Antiarteriosclerotic; Cardiant; Vasotropic; Neuroprotective; Nootropic; Neuroleptic; Antibacterial; Immunosuppressive; Hypertensive; Antirheumatic; Antiarthritic; Antiulcer; Gastrointestinal-Gen.; Antiinflammatory; Antidiabetic; Nephrotropic; Ophthalmological; Osteopathic; Virucide; Hepatotropic; Antigout; Antipruritic.
MECHANISM OF ACTION - Inducible **nitric oxide** synthase inhibitor.
(4R,5R)-5-ethyl-4-methyl-4,5-dihydro-1,3-thiazol-2-ylamine (Ia) inhibited iNOS with IC50 of 0.00655 mu M, with ratio iNOS/nNOS of 92.9 (nNOS = **nitric oxide** synthase), compared with IC50 of 0.0092 mu M for racemic trans-4,5-dimethyl-4,5- dihydro-1,3-thiazol-2-ylamine, with ratio iNOS/nNOS of 17.5.
USE - Used for treating arteriosclerosis, myocarditis, myocardial **disease**, cerebrovascular ischemic **disorders**, Alzheimer's **disease**, multiple sclerosis, septicemia, hypotension, chronic rheumatoid arthritis, arthritis deformans, gastric ulcer, duodenal ulcer, ulcerative colitis, diabetes, glomerulonephritis, uveitis, osteoporosis, pneumonia, hepatitis, ischemic reflux damage, post-transplant rejection, gout and **itching.**
ADVANTAGE - (I) Are highly selective for inducible **nitric oxide** synthase (iNOS) compared with constitutional **nitric oxide** synthase.
Dwg.0/0

FS CPI
FA AB; GI; DCN
MC CPI: B07-E01; B07-F01; B14-A01; B14-C02; B14-C03; B14-C09B; B14-D05; B14-E08; B14-F01B; B14-F02B; B14-F02D; B14-F07; B14-G02C; B14-K01; B14-N01; B14-N03; B14-N08; B14-N10; B14-N12; B14-N16; **B14-N17** ; B14-S01; B14-S04

L200 ANSWER 2 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2003-219908 [21] WPIX
DNC C2003-055834
TI Pharmaceutical composition useful for treating **pruritus** comprises at least one substance having an inhibitory activity of **nitric oxide** function.
DC B05
IN KURAISHI, Y; MIYAMOTO, T
PA (IKEM) IKEDA MOHANDO CO LTD; (KURA-I) KURAISHI Y; (MIYA-I) MIYAMOTO T
CYC 2
PI US 2002156129 A1 20021024 (200321)* 8p A61K031-198
JP 2002241308 A 20020828 (200321) 7p A61K045-00
ADT US 2002156129 A1 US 2001-982380 20011017; JP 2002241308 A JP 2001-44656 20010221
PRAI JP 2001-44656 20010221
IC ICM A61K031-198; A61K045-00
ICS A61K031-21; A61K031-22; A61K045-06; **A61P017-04**; A61P043-00
ICA A61K031-223
AB US2002156129 A UPAB: 20030328
NOVELTY - A pharmaceutical composition comprises at least one substance (I) having an activity of inhibiting an effect of **nitric oxide** (NO) in vivo and a carrier.
ACTIVITY - Antipruritic; Dermatological.
MECHANISM OF ACTION - **Nitrogen Oxide** Synthase (NOS) Inhibitor.
A rostral part (2 multiply 2 cm) of the back skin in each of ICR mice was shaved. Four days after the shaving the test was started: The skin was covered with a absorbent cotton impregnated (2 multiply 2 cm) with a mixture of acetone and a diethylether (1:1) for 15 seconds. The skin was wiped to remove superfluous solvent, then covered with an absorbent cotton impregnated with distilled water for 30 seconds, and wiped. This treatment for disrupting the barrier function of the skin was conducted twice daily at intervals of at least 8 hours for 5 days. On the next day each mouse was placed in each section of an acrylic acid resin cage divided into four sections. After the mice was acclimatized to be in the cage in unattended environment for 45 minutes, the test substance Nw-**nitro**-**L-arginine** methyl ester (**L-NAME**) or the control substance Nw-nitro-D-arginine methyl ester (D-**NAME**) in saline or saline alone was subcutaneously administered (10 mg/kg) at the location where the barrier-disrupting treatment was applied. The action of each mouse was filmed and recorded with a 8 mm video camera placed above the cage in such a manner that the 4 sections are in one scene or 2 hours. The **scratching** action was observed by playing back the video tape. The results of the test were as follows: The number of the **scratching** action in the group to which **L-NAME** was administrated, was reduced to 27.9 (+/-) 6.4 % of the control group to which saline was administrated. The number of the **scratching** action was 90.9 (+/-) 15.2 % in the group to which D-**NAME** was administered. The difference between **L-NAME** administrated group and D-**NAME** administrated group was statistically significant indicating that the inhibition of **L-NAME** is the result of the inhibition of NO synthesis, because the **scratching** action was only inhibited by **L-**

NAME and was not inhibited by the subcutaneous administration of equivalent amount of D-**NAME.**

USE - For treating noninflammatory and inflammatory **pruritus** (claimed).

ADVANTAGE - The composition is effective to **pruritus** which is associated with dry skin caused by the disruption of cutaneous barrier function and which is not associated with allergic inflammation, particularly effective to **pruritus** to which conventional histamine H1 antagonist is ineffective and provides overall improvement of quality of life or the relief of pains.

Dwg.0/2

FS CPI

FA AB; DCN

MC CPI: B04-H19; B06-D03; B06-D05; B06-F03; B07-D09; B07-F01; B10-A13A; B10-A17; B10-A20; **B14-N17**

L200 ANSWER 3 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 2002-683529 [74] WPIX

DNC C2002-193078

TI Cosmetic or dermatological preparations for reinforcing skin barrier function, e.g. to prevent dryness, containing agents inhibiting the onset of nitrogen monoxide synthase activity.

DC B05 D21 E19

IN DOERING, T; KOLBE, L; MUMMERT, C; MUNDT, C; SCHAUMANN, E; SCHREINER, V

PA (BEIE) BEIERSDORF AG

CYC 1

PI DE 10111054 A1 20020912 (200274)* 28p A61K007-48

ADT DE 10111054 A1 DE 2001-10111054 20010306

PRAI DE 2001-10111054 20010306

IC ICM A61K007-48

AB DE 10111054 A UPAB: 20021118

NOVELTY - The use of agents (I) which inhibit the onset of nitrogen monoxide (NO) synthase activity in warm-blood organisms is claimed in the production of cosmetic or dermatological preparations for reinforcing the barrier function of the skin.

ACTIVITY - Dermatological; antiallergic.

MECHANISM OF ACTION - NO synthase inhibitor; NO synthase expression inhibitor. (I) also stimulate the synthesis of collagen, hyaluronic acid, elastin and ceramide in the skin.

USE - (I) restore and/or reinforce the barrier function of the skin in protecting against environmental factors (e.g. dirt, chemicals or microorganisms) and loss of body components (e.g. water, natural lipids and electrolytes), and thus combat toxic or allergic reactions and dryness of the skin. (I) are also effective in the treatment and prophylaxis of deficiency, sensitivity or hypoactive conditions of the skin and exoskeleton.

ADVANTAGE - (I) are highly effective in restoring or maintaining the barrier function, moisture retention and physicochemical properties of the stratum corneum.

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B01-B02; B05-B01D; B06-D01; B06-D03; B06-D05; B06-D09; B06-E05; B07-D04C; B07-D05; B07-D09; B07-F02; B10-A01; B10-A06; B10-A07; B10-A12B; B10-A13D; B10-A17; B10-B02J; B10-C04E; B10-D03; B10-E02; B14-G02A; **B14-N17**; B14-N17C; B14-R01; D08-B09; E01; E05-K; E06-D01; E06-D03; E06-D05; E06-D09; E06-E05; E07-D04C; E07-D05; E07-D09; E07-F02; E10-A01; E10-A06A; E10-A07; E10-A12B2; E10-A13B2; E10-A17B; E10-B02D5; E10-C04E; E10-D03D; E10-E02U

L200 ANSWER 4 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2002-676043 [73] WPIX
DNC C2002-190711
TI Cosmetic or dermatological preparations for treating or preventing pigmentation disorders, e.g. freckles or liver spots, containing agents inhibiting the onset of nitrogen monoxide synthase activity.
DC B05 D21 E19
IN BLATT, T; KOLBE, L; MUMMERT, C; MUNDT, C; SCHAUMANN, E; WOLBER, R
PA (BEIE) BEIERSDORF AG
CYC 21
PI DE 10111050 A1 20020912 (200273)* 27p A61K007-48
WO 2002069910 A2 20020912 (200273) DE A61K007-00
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR
W: JP US
ADT DE 10111050 A1 DE 2001-10111050 20010306; WO 2002069910 A2 WO 2002-EP2120 20020228
PRAI DE 2001-10111050 20010306
IC ICM A61K007-00; A61K007-48
AB DE 10111050 A UPAB: 20021113
NOVELTY - The use of agents (I) which inhibit the onset of nitrogen monoxide (NO) synthase activity in warm-blood organisms is claimed in the production of cosmetic or dermatological preparations for the treatment and/or prophylaxis of pigmentation disorders.
ACTIVITY - Dermatological.
MECHANISM OF ACTION - NO synthase inhibitor; NO synthase expression inhibitor.
USE - (I) are useful for the treatment and/or prophylaxis of local hyper- or hypopigmentation conditions, e.g. liver spots, freckles, ephelides, scars or wound-, genetic- or aging-associated conditions; or for general, large-scale, cosmetic skin lightening.
ADVANTAGE - (I) are more effective and/or less harmful to the skin than prior art agents such as kojic acid, ascorbic acid, azelaic acid or their derivatives.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B01-B02; B06-D01; B06-D03; B06-D05; B06-D09; B06-E01; B06-E05; B07-D02; B07-D04C; B07-D05; B07-D06; B07-D09; B07-F03; B08-D03; B10-A01; B10-A06; B10-A07; B10-A12B; B10-A13A; B10-A13B; B10-A17; B10-B02J; B10-C04E; B10-D03; B10-E02; B14-C03; **B14-N17**; D08-B03; D08-B09; E01; E06-D01; E06-D03; E06-D05; E06-D09; E06-E01; E06-E05; E07-D02; E07-D04C; E07-D05; E07-D06; E07-D09; E07-F03; E08-D03; E10-A01; E10-A07; E10-A12B2; E10-A13A2; E10-A13B2; E10-A17B; E10-B02D5; E10-C04E; E10-D03; E10-D03D; E10-E02U

L200 ANSWER 5 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2002-668052 [72] WPIX
DNC C2002-187805
TI Cosmetic or dermatological preparations for combating inflammatory disorders or dryness of the skin, containing agents inhibiting the onset of nitrogen monoxide synthase activity.
DC B05 D21 E19
IN BLATT, T; KOLBE, L; KRUSE, I; MUMMERT, C; WOLBER, R
PA (BEIE) BEIERSDORF AG
CYC 1
PI DE 10111049 A1 20020912 (200272)* 27p A61K007-48
ADT DE 10111049 A1 DE 2001-10111049 20010306
PRAI DE 2001-10111049 20010306
IC ICM A61K007-48
ICS A61K007-40

AB DE 10111049 A UPAB: 20021108
NOVELTY - The use of agents (I) which inhibit the onset of nitrogen monoxide (NO) synthase activity in warm-blood organisms is claimed in the production of cosmetic or dermatological preparations for the prophylaxis and treatment of inflammatory skin conditions (including eczema) and/or protecting the skin against dryness determined by sensitivity.
ACTIVITY - Dermatological; antiinflammatory; antiallergic; antipruritic; antiseborrheic; vulnerary.
MECHANISM OF ACTION - NO synthase inhibitor; NO synthase expression inhibitor.
USE - (I) are useful for the treatment and/or prophylaxis of erythematous, inflammatory, allergic or autoimmune **disorders** of the skin, e.g. dermatosis, stinging, **itching**, acne or eczema; and for immunostimulation in the skin, e.g. to promote wound healing.
ADVANTAGE - (I) are highly effective in soothing sensitive or irritated skin.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B01-B02; B06-D01; B06-D03; B06-D05; B06-D09; B06-E01; B06-E05; B07-D02; B07-D04C; B07-D05; B07-D06; B07-D09; B07-F03; B08-D03; B10-A01; B10-A06; B10-A07; B10-A12C; B10-A13A; B10-A13B; B10-A17; B10-B02J; B10-C04E; B10-D03; B10-E02; B14-C03; B14-G02A; B14-N17A; B14-N17B; B14-N17C; B14-R02; D08-B03; D08-B09; E01; E06-D01; E06-D03; E06-D05; E06-D09; E06-E01; E06-E05; E07-D02; E07-D04C; E07-D05; E07-D06; E07-D09; E07-F03; E08-D03; E10-A01; E10-A06A; E10-A07; E10-A10; E10-A12B2; E10-A13A2; E10-A13B2; E10-A17B; E10-B02D5; E10-C04E; E10-D03; E10-E02U

L200 ANSWER 6 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2002-590911 [63] WPIX
DNC C2002-167330
TI Use of **nitric oxide** synthase inhibitors to reduce or prevent fine wrinkles.
DC B05 D21
IN FUJII, S; LERNER, E
PA (FUJI-I) FUJII S; (LERN-I) LERNER E; (GEHO) GEN HOSPITAL CORP
CYC 22
PI WO 2002062306 A1 20020815 (200263)* EN 15p A61K007-00
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR
W: JP
US 2002168325 A1 20021114 (200277) A61K007-42
US 2003207844 A1 20031106 (200374) A61K007-42
EP 1359885 A1 20031112 (200377) EN A61K007-00
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE TR
ADT WO 2002062306 A1 WO 2002-US2292 20020125; US 2002168325 A1 Provisional US 2001-264176P 20010125, US 2002-57247 20020125; US 2003207844 A1 Provisional US 2001-264176P 20010125, Div ex US 2002-57247 20020125, US 2003-406306 20030403; EP 1359885 A1 EP 2002-720854 20020125, WO 2002-US2292 20020125
FDT EP 1359885 A1 Based on WO 2002062306
PRAI US 2001-264176P 20010125; US 2002-57247 20020125; US 2003-406306 20030403
IC ICM A61K007-00; A61K007-42
ICS A61K007-44; A61K031-495; A61K031-655
AB WO 200262306 A UPAB: 20021001
NOVELTY - Method of preventing or treating a wrinkle by administration of a **nitric oxide** synthase (NOS) inhibitor to reduce or prevent the wrinkle is new.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a

kit for preventing wrinkles in a subject, and instructions on how to use the kit.

ACTIVITY - Antiaging.

In tests compounds were applied to the back skin of mice and their ability to prevent the formation of wrinkles caused by UVB exposure was evaluated. The NOS inhibitor L-NG-nitro-arginine methyl ester (**L-NAME**), at a concentration of 1% in 70% EtOH gave a total regular and fine wrinkle volume (in pixels) of 1.072,158 cf. 529,131 for an untreated control and 1,173,709 for UVB and 70% EtOH.

MECHANISM OF ACTION - None given in the source material.

USE - The method is preferably preventing or treating fine wrinkles caused by exposure to UVB radiation (claimed). It is for preventing or reducing symptoms of aging skin such as wrinkles, drying or cracking, particularly skin that has been exposed to the sun.

ADVANTAGE - **L-NAME** prevented the formation of fine wrinkles caused by UVB exposure.

Dwg.0/0

FS CPI
FA AB; DCN
MC CPI: B14-D08; **B14-N17**; B14-R04; D08-B09A1; D08-B09A3

L200 ANSWER 7 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2001-503418 [56] WPIX
DNC C2001-151470
TI Use of one or more **nitric oxide** synthase inhibitors e.g. **nitroarginine** in cosmetic and dermatological compositions for the treatment and prevention of intrinsic and/or extrinsic skin ageing.
DC B05 D21
IN HINZE, C; KRUSE, I; SCHOENROCK, U; WILKE, J
PA (BEIE) BEIERSDORF AG
CYC 1
PI DE 10000840 A1 20010719 (200156)* 8p A61K007-48
ADT DE 10000840 A1 DE 2000-10000840 20000112
PRAI DE 2000-10000840 20000112
IC ICM A61K007-48
AB DE 10000840 A UPAB: 20011001

NOVELTY - Use of one or more **nitric oxide** synthase inhibitors in cosmetic and dermatological compositions for the treatment and prevention of intrinsic and/or extrinsic skin ageing.

ACTIVITY - Dermatological; Antipsoriatic; Antiinflammatory; Antipruritic

MECHANISM OF ACTION - Antioxidant; Collagen-Agonist; Hyaluronate-Agonist

USE - The compositions are useful as antioxidants in cosmetic and dermatological compositions for the treatment and prevention of intrinsic and/or extrinsic skin ageing. They are especially useful for the treatment of photodermatosis, preferably polymorphic light dermatosis (Mallorca acne), as well as deficiency-, sensitivity- and hypoactivity-associated skin **disorders**, wrinkles/blemishes, environmental skin damage (caused by cigarette smoke, smog, and free radicals), pigmentation **disorders**, **itching**, dry skin, hair loss, atopic eczema, seborrheic eczema, psoriasis and vitiligo. The compositions stimulate synthesis of collagen, hyaluronic acid, elastin and intracellular DNA, and improve the skin's repair mechanism. They can be administered as part of laser therapy, to prevent blemishes and scarring.

ADVANTAGE - There is a reduced incidence of side effects.

Dwg.0/0

FS CPI
FA AB; DCN

MC CPI: B10-A17; B14-D05A; B14-L01; **B14-N17**; B14-R01; B14-R02; B14-S08; D08-B09A

L200 ANSWER 8 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2001-234905 [24] WPIX
DNC C2001-070327
TI New compounds including drug groups used for treating oxidative stress and/or endothelial disorders of moderate intensity.
DC B05
IN DEL SOLDATO, P; DEL SOLDATA, P
PA (NICO-N) NICOX SA
CYC 83
PI WO 2001012584 A2 20010222 (200124)* EN 93p C07C219-14
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AU BA BB BG BR CA CN CR CU CZ DM EE GD GE HR HU ID IL IN IS JP KP KR LC LK LR LT LV MA MG MK MN MX NO NZ PL RO SG SI SK TR TT UA US UZ VN YU ZA
AU 2000065670 A 20010313 (200134) C07C219-14
BR 2000013264 A 20020416 (200234) C07C219-14
NO 2002000623 A 20020409 (200238) C07C000-00
KR 2002032552 A 20020503 (200270) C07D499-68
EP 1252133 A2 20021030 (200279) EN C07C219-14
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
IT 1314184 B 20021206 (200317) A61K031-00
JP 2003515526 W 20030507 (200331) 116p C07C203-04
HU 2002003939 A2 20030328 (200333) C07C219-14
ZA 2002000628 A 20030625 (200348) 110p C07C000-00
CN 1433396 A 20030730 (200365) C07C219-14
MX 2002001519 A1 20030701 (200366) A61K031-21
ADT WO 2001012584 A2 WO 2000-EP7225 20000727; AU 2000065670 A AU 2000-65670 20000727; BR 2000013264 A BR 2000-13264 20000727, WO 2000-EP7225 20000727; NO 2002000623 A WO 2000-EP7225 20000727, NO 2002-623 20020208; KR 2002032552 A KR 2002-701883 20020209; EP 1252133 A2 EP 2000-953102 20000727, WO 2000-EP7225 20000727; IT 1314184 B IT 1999-MI1817 19990812; JP 2003515526 W WO 2000-EP7225 20000727, JP 2001-516885 20000727; HU 2002003939 A2 WO 2000-EP7225 20000727, HU 2002-3939 20000727; ZA 2002000628 A ZA 2002-628 20020123; CN 1433396 A CN 2000-814049 20000727; MX 2002001519 A1 WO 2000-EP7225 20000727, MX 2002-1519 20020211
FDT AU 2000065670 A Based on WO 2001012584; BR 2000013264 A Based on WO 2001012584; EP 1252133 A2 Based on WO 2001012584; JP 2003515526 W Based on WO 2001012584; HU 2002003939 A2 Based on WO 2001012584; MX 2002001519 A1 Based on WO 2001012584
PRAI IT 1999-MI1817 19990812
IC ICM A61K031-00; A61K031-21; C07C000-00; C07C203-04; C07C219-14; C07D499-68
ICS A61K031-221; A61K031-222; A61K031-235; A61K031-365; A61K031-366; A61K031-43; A61K031-4365; A61K031-437; A61K031-454; A61K031-473; A61K031-496; A61K031-663; A61K031-704; A61K038-00; A61P001-02; A61P003-06; A61P003-10; A61P007-02; A61P009-08; A61P009-12; A61P011-06; A61P011-08; A61P011-10; A61P011-12; A61P019-08; A61P025-02; A61P025-28; A61P029-00; A61P031-04; A61P031-12; A61P035-00; A61P037-08; A61P043-00; C07C219-22; C07C219-24; C07C219-30; C07C229-42; C07C233-25; C07D213-00; C07D219-10; C07D295-08; C07D307-80; C07D309-30; C07D333-00; C07D401-12; C07D471-04; C07D495-00; C07D495-04; C07D499-48; C07F009-38; C07H015-252
AB WO 200112584 A UPAB: 20030113
NOVELTY - New compounds (I) including drug groups are new.

DETAILED DESCRIPTION - Compounds of formula A-B-N(O)s (I) are new.
s = 1 or 2, preferably 2;
A = R-T1;
R = a drug group;
T1 = (CO)t or (X)t;
X = O, S or NR1c;
t, t' = 0 or 1;
provided that when t = 1 when t' = 0 and t = 0 when t' = 1;
B = TB-X2-O;
TB = CO when t = 0 or X when t' = 0;
X2 = a bivalent group such that the corresponding precursor TB-X2-OH of B does not meet test 5 and meets test 4A and TB = CO and t = 0, with the free valence of TB saturated with OZ or ZI-N(ZII) or TB = X and t' = 0 and the free valence of TB is saturated with H;
Z = H or R1a;
R1a = 1-10 (preferably 1-5)C alkyl and
ZI, ZII = a group Z;
provided that the drug A = R-T1, where the free valence is saturated when t' = 0, with OZ or ZI-N(ZII) and when t = 0 with X-Z meets at least one of tests 1-3.

Test 1 (NEM) is a test carried out in vivo on 4 groups of rats (each group containing 10 rats), the controls (2 groups) and the treated (2 groups) of which one group of the controls and one group of the treated respectively are administered with one dose of 25 mg/kg subcutaneously N-ethylmaleimide (NEM). The controls are treated with the carrier and the treated groups with carrier and drug A = R-T1 with saturated free valence. The drug is administered at a dose equivalent to the maximum dose tolerated by the rats that did not receive NEM. The drug can be used to prepare (I) when the group treated with NEM, carrier and drug shows gastrointestinal damage or in the group treated with NEM, carrier and drug are observed gastrointestinal damage greater than that of the group treated with carrier or of the group treated with the carrier and NEM.

Test 2 (CIP) is an in vitro test where human endothelial cells from the umbilical vein are harvested under standard conditions, then divided into 2 groups (each replicated 5 times), of which one is treated with a mixture of the drug 10-4 concentration in culture medium and the other group with carrier. Then cumene hydroperoxide (CIP) having 5 mM concentration in the culture medium is added to each group. The drug can be use to prepare (I) when a statistically significant inhibition of the apoptosis induced by CIP is not obtained with p less than 0.01 with respect to the group treated with carrier and CIP.

Test 3 (**l-NAME**) is an in vivo test carried out on 4 groups of rats (each containing 10 rats) for 4 weeks and receiving drinking water, the controls (2 groups) and the treated (2 groups), of which 1 group of controls and of treated respectively receive in the above weeks water containing **N- omega -nitro-L-arginine** methyl ester (**L-NAME**) at a concentration of 400 mg/l. Controls in the 4 weeks are administered with carrier and the treated in the 4 weeks with carrier and drug, each once a day. The drug is administered at the maximum dose tolerated by the group of rats not pretreated with **L-NAME.** After 4 weeks, water supply is stopped for 24 hours and then the rats are sacrificed. Blood pressure is determined 1 hour before sacrifice. After sacrifice, the plasma glutamic pyruvic transaminase (GPT) is determined and the gastric tissue is examined. The drug can be used to prepare (I) when in group treated with **L-NAME**, carrier and drug, greater hepatic damage and/or cardiovascular damage are found in comparison respectively with the group treated with the carrier or carrier and drug or carrier and **L-NAME.**

Test 4A met by the compound precursor B is an in vitro test in which

part of an erythrocyte suspension kept at 4 deg. C for 4 days and isolated from Wistar male rats and suspended in physiological solution buffered at pH 7.4 with phosphate buffer, is centrifuged at 1000 rpm for 5 minutes. 0.1 ml Centrifuged erythrocytes are diluted with sodium phosphate buffer pH 7.4 at 50 ml. Aliquots of 3.5 ml are taken and incubated at 37degC in the presence of cumene hydroperoxide at a concentration of 270 mu M and the suspension turbidity determined at 710 nm at intervals of 30 minutes to establish the time (Tmax) at which occurs the maximum turbidity that corresponds to the maximum amounts of cells lysed by cumene hydroperoxide (haemolysis assumed to be 100%). Alcoholic solutions of the compounds precursors of B are added to 3.5 ml aliquots of the dilutes suspension of centrifuged erythrocytes to give a final concentration of 2 mm of the precursor of B. Resulting suspension is preincubated for 30 minutes. Cumene hydroperoxide is added to give the same above indicated final concentration and at Tmax is determined the percentage of haemolysis inhibition in the sample from the ratio, multiplied by 100, between absorbance of sample containing erythrocytes, precursor of B and cumene hydroperoxide respectively and that of sample containing erythrocytes and cumene hydroperoxide. Precursors of B meet the test if they inhibit haemolysis induced by cumene hydroperoxide by more than 15%.

Test 5 is an analytical determination carried out by adding aliquots of 10-4 M methanol solutions of precursor B or B1 or of C = Tc-Y-H, having the free valence saturated, to solution formed by admixing 2 mM solution of deoxyribose in water with 100 mM phosphate buffer and 1 mu M FeII(NH4)2(SO4)2. After thermostating at 37 deg. C for 1 hour, aliquots of aqueous solutions of trichloroacetic acid (2.8%) and of thiobarbituric acid (0.5M) are added and heating is effected at 100 deg. C for 15 minutes. Absorbance of tested solutions is read at 532 nm. Inhibition induced by precursor B or B1 or C = Tc-Y-H in the confront of radical production by FeII is calculated as a percentage by using (1-As/Ac) x 100.

As and Ac are respectively absorbance values of solution containing tested compound and iron salt and that of solution containing iron salt. Test 5 is met when inhibition percentage is at least 50%.

In (I), when X2 of B is 1-20C alkylene or 5-7C cycloalkylene (optionally substituted), the drugs of formula A = R-T1 with free valence saturated, do not belong to drugs used in incontinence, antithrombotic drugs (ACE inhibitors), prostaglandins and anti-inflammatory drugs (NSAIDs and corticosteroids), but not excluding paracetamol and sulindac.

N.B. The definitions given in the specification are not clear.

ACTIVITY - Antioxidant; cardiant; vasotropic; hypotensive; cerebroprotective; antiarteriosclerotic; antiarthritic; anti-inflammatory; neuroprotective; dermatological; antibacterial.

MECHANISM OF ACTION - None given.

USE - Used for treating oxidative stress and/or endothelial dysfunctions of moderate intensity, which cause myocardial and vascular ischemia, hypertension, stroke, arteriosclerosis, rheumatoid arthritis and connected inflammatory diseases, asthma and connected inflammatory diseases, ulcerative and non ulcerative dyspepsias, intestinal inflammatory diseases, Alzheimer's disease, impotence, incontinence, eczema, neurodermatitis, acne and infectious diseases.

ADVANTAGE - (I) Have higher efficacy and lower toxicity.

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B02-T; B05-B01G; B06-A01; B06-D05; B06-D08; B06-D13; B06-F03; B07-A02B; B07-A04; B07-B01; B07-B03; B07-D01; B07-D02; B07-D05; B07-E01; B07-F01; B10-A05; B10-B03B; B10-E04C; B14-A01; B14-C03; B14-C09; B14-F01; B14-F02; B14-F02B; B14-F07; B14-J01; **B14-N17**; B14-S08

L200 ANSWER 9 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2001-183074 [18] WPIX
CR 1997-011834 [01]; 2002-025885 [03]; 2002-443183 [47]; 2002-690385 [74]
DNC C2001-054686
TI Diagnosis and treatment of e.g. irritable bowel syndrome, chronic fatigue syndrome, depression, or Crohn's disease comprises detecting the presence of small intestinal bacterial overgrowth in a subject having at least one associated symptom.
DC B04 B05
IN LIN, H C; PIMENTAL, M; PIMENTEL, M
PA (CEDA-N) CEDARS SINAI MEDICAL CENT; (LINH-I) LIN H C; (PIME-I) PIMENTEL M
CYC 95
PI WO 2001011077 A2 20010215 (200118)* EN 43p C12Q001-00
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2000065383 A 20010305 (200130) C12Q001-00
EP 1200828 A2 20020502 (200236) EN G01N033-569
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
US 2003031625 A1 20030213 (200314) A61M036-14
ADT WO 2001011077 A2 WO 2000-US22030 20000811; AU 2000065383 A AU 2000-65383 20000811; EP 1200828 A2 EP 2000-952739 20000811, WO 2000-US22030 20000811; US 2003031625 A1 Div ex US 1999-374142 19990811, US 2002-107240 20020326
FDT AU 2000065383 A Based on WO 2001011077; EP 1200828 A2 Based on WO 2001011077
PRAI US 1999-374142 19990811; US 2002-107240 20020326
IC ICM A61M036-14; C12Q001-00; G01N033-569
ICS A61K049-00; A61K051-00; C12Q001-04
AB WO 200111077 A UPAB: 20030227
NOVELTY - Diagnosis of irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, depression, attention deficit/hyperactivity disorder, an autoimmune disease or Crohn's disease comprises detecting the presence of small intestinal bacterial overgrowth in a subject having at least one symptom associated with the diagnosis of one of these diseases.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(A) treatment of irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, depression, attention deficit/hyperactivity disorder, an autoimmune disease or Crohn's disease comprising detecting the presence of small intestinal bacterial overgrowth in a subject having at least one symptom associated with the diagnosis of one of these diseases, and eradicating the bacterial overgrowth; and

(B) a kit for the diagnosis of irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, depression, attention deficit/hyperactivity disorder, an autoimmune disease or Crohn's disease comprising at least one breath sampling container, a pre-measured amount of substrate and instructions for the user for detecting the presence of small intestinal bacterial overgrowth in a subject having at least one symptom associated with the diagnosis of one of these diseases.

ACTIVITY - Antiinflammatory; antidepressant; nootropic; tranquilizer; immunosuppressive; neuroprotective; dermatological.

Thirty subjects had previously received a diagnosis of chronic fatigue syndrome. Of these 30, 21 had small intestinal bacterial overgrowth (SIBO) as indicated by lactulose breath hydrogen testing (LBHT). Four out of the nine who did not have SIBO indicated had already received antibiotics. After treatment with neomycin (500 mg, twice daily

for 10 days), 9 of the subjects with SIBO returned for LBHT and questionnaire. LBHT showed that all nine subjects experienced at least partial eradication of SIBO, and symptoms of bloating and fatigue were substantially improved.

MECHANISM OF ACTION - None given.

USE - The methods are used for the diagnosis and treatment of irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, depression, attention deficit/hyperactivity disorder, an autoimmune disease (e.g. multiple sclerosis or systemic lupus erythematosus) or Crohn's disease (claimed).

ADVANTAGE - The method diagnoses and treats the underlying causal factor of the diseases.

Dwg.0/2

FS CPI

FA AB; DCN

MC CPI: B02-Z; B04-F10; B05-C04; B05-C08; B06-D02; B07-A02B; B07-D09; B10-A07; B10-J02; B12-K04A; B14-A01; B14-E10C; B14-G02D; B14-J01A; **B14-N17**; B14-S01

L200 ANSWER 10 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 2001-061287 [07] WPIX

CR 1998-322324 [28]

DNC C2001-016915

TI **Nitric oxide** synthase inhibitors are useful for increasing the population of hematopoietic stem cells capable of undergoing normal hematopoiesis, differentiation and maturation and for regenerating tissue.

DC B05

IN CLINE, H; ENIKOLOPOV, G; KUZIN, B A; MICHURINA, T; PEUNOVA, N I

PA (COLD-N) COLD SPRING HARBOR LAB

CYC 94

PI WO 2000071112 A1 20001130 (200107)* EN 67p A61K031-131

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2000052752 A 20001212 (200115)

EP 1185258 A1 20020313 (200225) EN A61K031-131

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

JP 2003500355 W 20030107 (200314) 70p A61K035-28

MX 2001011386 A1 20020301 (200362) A61K031-131

ADT WO 2000071112 A1 WO 2000-US13685 20000518; AU 2000052752 A AU 2000-52752 20000518; EP 1185258 A1 EP 2000-937605 20000518, WO 2000-US13685 20000518; JP 2003500355 W JP 2000-619419 20000518, WO 2000-US13685 20000518; MX 2001011386 A1 WO 2000-US13685 20000518, MX 2001-11386 20011108

FDT AU 2000052752 A Based on WO 2000071112; EP 1185258 A1 Based on WO 2000071112; JP 2003500355 W Based on WO 2000071112; MX 2001011386 A1 Based on WO 2000071112

PRAI US 1999-315929 19990520

IC ICM A61K031-131; A61K035-28

ICS A61K031-155; A61K031-198; A61K031-223; A61P007-00; A61P007-06; C12N005-06

ICI C12N005-06; C12R001:91

AB WO 200071112 A UPAB: 20030928

NOVELTY - A method for increasing the population of hematopoietic stem cells capable of undergoing normal hematopoiesis, differentiation and maturation in hematopoietic tissue comprises treating the tissue with

multiple doses of **nitric oxide** synthase inhibitors.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for:

(i) a method for treating a mammal to increase the population of hematopoietic stem cells capable of undergoing normal hematopoiesis, differentiation and maturation comprising administration of multiple doses of a **nitric oxide** synthase inhibitor;

(ii) a method for treating a mammal to increase the population of hematopoietic stem cells capable of undergoing normal hematopoiesis, differentiation and maturation comprising transplanting hematopoietic tissue which has been treated with multiple doses of **nitric oxide** synthase inhibitors into the mammal;

(iii) a method of increasing a population of progenitor blood cells capable of undergoing normal hematopoiesis, differentiation and maturation comprising treating the cells with multiple doses of **nitric oxide** synthase inhibitors;

(iv) a method of increasing a population of dividing cells comprising treatment with multiple doses of **nitric oxide** inhibitors;

(v) a method of decreasing a population of cells in S phase and inducing differentiation of cells comprising treatment with multiple doses of **nitric oxide** enhancers;

(vi) a method of regenerating tissue comprising treating the tissue with multiple doses of a **nitric oxide** inhibitor; and

(vii) a method of repopulating an organ having normally non-dividing cells comprising treatment with multiple doses of **nitric oxide** inhibitors.

USE - The method is useful for increasing the population of hematopoietic stem cells capable of undergoing normal hematopoiesis, differentiation and maturation in hematopoietic tissue and for regenerating tissue.

Dwg.0/0

FS CPI
FA AB; DCN
MC CPI: B04-H06; B08-D02; B10-A17; B14-D08; **B14-N17**

L200 ANSWER 11 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2000-681104 [67] WPIX
DNC C2000-207281
TI Use of **nitric oxide** scavengers or inhibitors to improve permeability barrier function of skin and/or to improve structure or growth of hair.
DC B05 D21
IN DOERING, T; MAX, H; SANDHOFF, K; SAUERMANN, G; SCHOENROCK, U; SCHREINER, V
PA (BEIE) BEIERSDORF AG
CYC 25
PI EP 1046392 A2 20001025 (200067)* DE 13p A61K007-48
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
DE 19918750 A1 20001026 (200067) A61K007-48
ADT EP 1046392 A2 EP 2000-108526 20000419; DE 19918750 A1 DE 1999-19918750 19990424
PRAI DE 1999-19918750 19990424
IC ICM A61K007-48
ICS A61K007-00; A61K007-06; A61K007-40
AB EP 1046392 A UPAB: 20001223
NOVELTY - Substances (I) that bind **nitric oxide** (NO) or NO radicals or inhibit NO synthesis or inhibit the effects of NO and NO radicals are used topically to improve the permeability barrier function of the skin and/or to improve the structure or growth of hair.

ACTIVITY - Dermatological.

MECHANISM OF ACTION - **Nitric oxide** inhibitor.
USE - (I) are especially useful in cosmetic or dermatological compositions for the care or cleansing of dry, stressed or aged skin, for the treatment and prophylaxis of dry and stressed skin states and their sequelae and for the treatment and prophylaxis of neurodermatitis and non-eczematous atopic dermatitis.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B10-A17; **B14-N17**; B14-R01; D08-B09A

L200 ANSWER 12 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2000-171083 [15] WPIX
CR 1993-288089 [36]; 1997-319538 [29]; 1998-427937 [36]; 1998-582508 [49]; 1999-008650 [01]; 2000-171084 [15]; 2000-182219 [16]
DNN N2000-127157 DNC C2000-053204
TI Stimulating vascularization for e.g. promoting wound healing by contacting site with matrix comprising gelatin and **nitric oxide** inhibitor.
DC B03 B05 C02 C03 P32 P34
IN USALA, A; KLANN, R C
PA (ENCE-N) ENCELLE INC; (USAL-I) USALA A
CYC 87
PI WO 2000002596 A1 20000120 (200015)* EN 28p A61L015-32
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW
AU 9950952 A 20000201 (200028)
EP 1094849 A1 20010502 (200125) EN A61L015-32
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
US 6261587 B1 20010717 (200142) A61F002-02
US 2001007658 A1 20010712 (200143) C12N005-00
US 2001010826 A1 20010802 (200147) A61K009-06
AU 757968 B 20030313 (200328) A61L015-32
ADT WO 2000002596 A1 WO 1999-US15614 19990709; AU 9950952 A AU 1999-50952 19990709; EP 1094849 A1 EP 1999-935483 19990709, WO 1999-US15614 19990709; US 6261587 B1 CIP of US 1998-113437 19980710, US 1999-337959 19990622; US 2001007658 A1 CIP of US 1992-841973 19920224, CIP of US 1994-300429 19940902, CIP of US 1995-568482 19951207, Div ex US 1998-113437 19980710, US 2001-758676 20010111; US 2001010826 A1 CIP of US 1992-841973 19920224, CIP of US 1994-300429 19940902, CIP of US 1995-568482 19951207, CIP of US 1998-113437 19980710, Div ex US 1999-337959 19990622, US 2001-766330 20010119; AU 757968 B AU 1999-50952 19990709
FDT AU 9950952 A Based on WO 2000002596; EP 1094849 A1 Based on WO 2000002596; US 2001007658 A1 CIP of US 5834005, Div ex US 6231881; US 2001010826 A1 CIP of US 5834005, CIP of US 6231881; AU 757968 B Previous Publ. AU 9950952, Based on WO 2000002596
PRAI US 1999-337959 19990622; US 1998-113437 19980710; US 1992-841973 19920224; US 1994-300429 19940902; US 1995-568482 19951207; US 2001-758676 20010111; US 2001-766330 20010119
IC ICM A61F002-02; A61K009-06; A61L015-32; C12N005-00
ICS A61L015-44; A61L031-00; A61L033-00; C12N005-02
AB WO 200002596 A UPAB: 20030501
NOVELTY - Stimulating vascularization at sites in mammals comprises contacting the site with a matrix comprising gelatin and **nitric oxide** inhibitor.

ACTIVITY - Dermatological; antiinflammatory; antidiabetic; antiarteriosclerotic; cardiant; neuroprotective; antiarthritic; respiratory; CNS.

The ability of test matrix to stimulate blood vessels in fibrous capsule was compared to Matrigel (RTM: laminin, collagen IV, entactin, heparan sulfate proteoglycan matrix) with or without basic fibroblast growth factor (bFGF) or vascular endothelial growth factor (VEGF) when applied around polycarbonate devices intramuscularly in rats. Devices surrounded by these materials or no material were removed from some rats at day 21 and some rats at day 50. Capsule thickness and vascular density of the capsule were evaluated. Only the test matrix stimulated new blood vessel growth between 21 and 50 days post-injection. While all other groups stimulated initial new blood vessel growth up to 21 days, a diminution in both blood vessel number and fibrous capsule thickness was documented as mature scar tissue was formed. In addition, matrix treated animal did not show the immune cell/inflammatory response observed in the bFGF- or VEGF-treated animals.

MECHANISM OF ACTION - Fibroblast scaffold; fibroblast stimulator; **nitric oxide** inhibitor; **nitric oxide** scavenger; endothelial proliferation stimulator.

USE - Used to stimulate vascularization at sites in mammals, to treat vascular disorders, to prevent immune reactions at anatomic sites, to promote wound healing and to prevent scar tissue formation at anatomic sites (claimed) including sites of chronic inflammation, atherosclerosis and sites where transplant of cells and/or organs will be placed such as the muscles and body cavities (peritoneal, abdominal). The method is also used to improve vascularization and to stimulate and maintain vascularization at predetermined sites in host organisms and in host organisms that would benefit from increased blood supply including those with gangrene, diabetes, poor circulation, atherosclerosis, arteriosclerosis, coronary artery disease, aortic aneurysm, arterial disease of the lower extremities, cerebrovascular disease, diseased or hypoxic hearts, particularly where vessels to the heart are obstructed, other organs with arterial sclerosis (ischemic bowel disease, cerebrovascular disease, vascular impotence), diabetic peripheral vascular disease, cerebral ischemia, ischemic heart disease and Raynaud's phenomenon and to improve kidney function.

The method is also used in transplant therapies to prepare transplant sites for organ or tissue transplants e.g. pancreas, kidney, heart, lung and liver transplants. The matrix is injected into ischemic myocardium to enhance development of collaterals, accelerate healing of necrotic tissue and prevent infarct expansion and cardiac dilatation. The method is also used to improve circulation post-stroke or post-heart attack, to prevent or reduce inflammatory response in chronic inflammatory diseases such as rheumatoid arthritis, atherosclerosis, tuberculosis, chronic lung diseases, autoimmune diseases and lupus erythematosus and to administer cells to replace or inject into the central nervous system to treat e.g. Parkinson's disease, Huntington's disease, Alzheimer's disease, bipolar disease and schizophrenia. The matrix is used with donor cells such as genetically modified donor cells including fibroblasts, neurons, glial cells, keratinocytes, hepatocytes, connective tissue cells, ependymal cells, chromaffin cells and other mammalian cells susceptible to genetic manipulation and grafting and to prepare vascularized bed for transplantation of embryos in in vitro fertilization and for superficial wound healing e.g. in skin ulcers, burn areas, ulcers secondary to peripheral vascular disease or other tissue damage. The matrix is applied to surgical breast implants to minimize painful adhesions. The method is used for humans, dogs, cows, pigs, sheep, cats and horses.

ADVANTAGE - The matrix stimulates local blood vessel growth within thin, fibrous capsule sheet and stimulates or enhances vascularization

without immune cell stimulation at the site resulting in long-term functional vascularity.
Dwg.0/4
FS CPI GMPI
FA AB; DCN
MC CPI: B04-N02; B10-A09B; B10-A17; B14-A01B1; B14-C03; B14-C09B; B14-E10; B14-F01B; B14-F02D1; B14-F07; B14-G02; B14-G02D; B14-J01A3; B14-J01A4; B14-J01B3; B14-K01; B14-L06; B14-N10; **B14-N17**; B14-S04; C04-N02; C10-A09B; C10-A17; C14-A01B1; C14-C03; C14-C09B; C14-F01B; C14-F07; C14-G02; C14-G02D; C14-J01A3; C14-J01A4; C14-J01B3; C14-K01; C14-L06; **C14-N17**

L200 ANSWER 13 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1996-502621 [50] WPIX
DNC C1996-157292
TI Dermatological drug for external use - comprising **nitric oxide** synthase inhibitors.
DC B05 D21 E16
PA (SHIS) SHISEIDO CO LTD
CYC 1
PI JP 08259435 A 19961008 (199650)* 5p A61K007-48
ADT JP 08259435 A JP 1995-91816 19950324
PRAI JP 1995-91816 19950324
IC ICM A61K007-48
ICS A61K007-00; A61K031-22
AB JP 08259435 A UPAB: 19961211
Dermatological drug for external use comprises one or more **nitric oxide** synthase inhibitors.
Nitric oxide synthase inhibitor is L-**nitroarginine** methyl ester. The drug comprises at least 0.8 weight % **nitric oxide** synthase inhibitor in the base.
USE/ADVANTAGE - The drug is used for improvement of ruddy face (claimed). The drug improves ruddy face and is highly safe.
In an example, vanishing cream comprised 5.0 weight % stearic acid, 4.0 weight % stearyl alcohol, 8.0 weight % butyl alcohol stearic acid ester, 2.0 weight % glycerin monostearic acid ester, 1.0 weight % L-**nitroarginine** methyl ester, 10.0 weight % propylene glycol, 4.0 weight % glycerin, 0.2 weight % sodium potassium, antiseptic, antioxidant, fragrance and ion-exchange water.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B10-A17; **B14-N17**; D08-B09A; E10-B02D7

L200 ANSWER 14 OF 14 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1995-206686 [27] WPIX
DNC C1995-095752
TI **Nitric oxide** synthase inhibitors in auto-immune disease treatment - comprises guanidine or arginine derivs..
DC B05
IN WEINBERG, J B; WEINBERG, J
PA (UYDU-N) UNIV DUKE MEDICAL CENT
CYC 60
PI WO 9513805 A1 19950526 (199527)* EN 27p A61K031-22
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MW NL NO NZ PL PT RO RU SD SE SI SK TJ TT UA US UZ VN

AU 9512099 A 19950606 (199538) A61K031-22
EP 729356 A1 19960904 (199640) EN A61K031-22
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
JP 09508891 W 19970909 (199746) 27p A61K045-00
ADT WO 9513805 A1 WO 1994-US13239 19941117; AU 9512099 A AU 1995-12099 19941117; EP 729356 A1 WO 1994-US13239 19941117, EP 1995-903124 19941117; JP 09508891 W WO 1994-US13239 19941117, JP 1995-514592 19941117
FDT AU 9512099 A Based on WO 9513805; EP 729356 A1 Based on WO 9513805; JP 09508891 W Based on WO 9513805
PRAI US 1993-153469 19931117
REP 15Jnl.Ref; AU 5453490; EP 547558; WO 9104023; WO 9313055
IC ICM A61K031-22; A61K045-00
ICS A61K031-155; A61K031-19; A61K031-195; A61K031-215
AB WO 9513805 A UPAB: 19950712
Treating or preventing autoimmune diseases in a patient, comprising admin. of a **nitric oxide** synthase inhibitor (NOSI), or a **nitric oxide** scavenger, is new.

The NOSI is N-G-amino=L-arginine, N-G-methyl-L-arginine (NMMA), N-G-**nitro-L-arginine** and its methyl ester, N-G-iminoethyl-L-ornithine, or aminoguanidine, most pref. NMMA.

Examples of **nitric oxide** scavengers are haemoglobin and the cobalamins.

USE - **Nitric oxide** has potent proinflammatory actions, increasing vascular permeability, and mediates pain in inflammation. Used for treating autoimmune diseases e.g. rheumatoid arthritis, insulin dependent diabetes mellitus, systemic lupus erythematosus, and glomerulonephritis.

Admin. is enteral (oral), parenteral, or topical. Doses are not given.

Dwg.0/1
FS CPI
FA AB; DCN
MC CPI: B04-H03; B10-A03; B10-A05; B10-A17; B14-C03; B14-C09B; B14-D10; B14-F09; B14-G02D; B14-L06; B14-L08; B14-N10; B14-N14; **B14-N17**

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